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STUDIES ON TUMOR METASTASIS†

3. The Distribution of Metastases In Carcinoma of the Large Intestine

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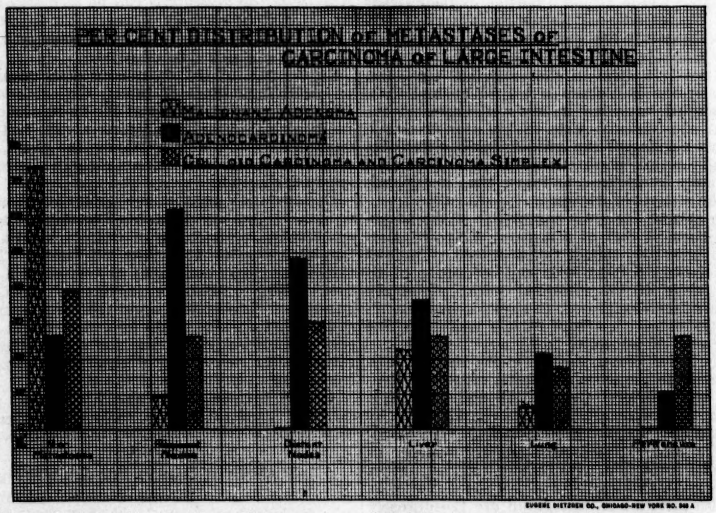
WE have grouped together for study of their metastases 156 cases of cancer of the large bowel* derived from the post mortem material of the New England Deaconess, New England Baptist and Pondville Hospitals. The cases of cancer of the rectum are by far the most numerous, 91; 90 cases were carcinoma of various types, and one case was a melanotic sarcoma. In the sigmoid 19 cases were encountered, all carcinoma. In the remainder of the colon, other than the cecum, there were 21 cases, and in the cecum 25 cancers in 24 cases (one case of multiple malignancy, a colloid carcinoma and an adenocarcinoma). Comparison with cancers of the small intestine shows the usual striking disproportion, the cases in the same autopsy material numbering only six, distributed as follows: one adenocarcinoma of the duodenum, two adenocarcinomas and one leiomyosarcoma of the jejunum, one leiomyosarcoma of the ileum, and one lymphosarcoma involving much of the small intestine. The high proportion of mesenchymal tumors in the small intestine is also striking.


PER CENT DISTRIBUTION OF METASTASES OF CARCINOMA OF LARGE INTESTINE
ADENOCARCINOMA
CYLINDRICAL CARCINOMA AND CARCINOMA SIMPLEX
EUGENE DIETZGEN CO., CHICAGO-NEW YORK NO. 340 A

We have endeavored to grade the carcinomas on the basis of their histologic appearance, grouping them on the basis of the degree of resemblance which they show to the normal mucosa of the large intestine. The most highly differentiated group, the malignant adenomas, parallels the Grade I cancers of Rankin and Broders[1] and of Stewart and Spies[2].

The most important criterion for inclusion in the group of malignant adenomas is the maintenance of a clear-cut alveolar structure with rare or no cell masses and with most of the

*Many of the cases were derived from the service of Dr. Daniel Fiske Jones, for whose interest and advice we are deeply grateful.

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cells of the columnar type. Mucous secretion may be fairly plentiful but when it occurs the secretion is all within the lumina of the alveoli rather than into the stroma, as in the case of the so-called colloid carcinoma. Mitoses may be very frequent, but are practically all of normal type. The stroma varies considerably, at times virtually none, the tumor infiltrating the submucosa, the muscularis and even the serosa. At other times a considerable amount of stroma is formed.

The gross appearance of the malignant adenoma is usually that of a large, exophytic mass, usually pedunculated, although sometimes sessile, and only in advanced cases showing marked infiltration, so far as gross evidence goes, of the deeper layers of the muscularis or serosa. Annular growth when present is a late manifestation.

The second group is made up of the adenocarcinomas. This includes all of Rankin and Broders' Grade II and a portion of their Grade III. In the adenocarcinoma there is present in at least twenty-five per cent of the tumor definite glandular formation. At times the glands are nearly normal in character, but more frequently the epithelium is heaped up and there is marked variation in size and shape of the cells. In various parts of the tumor considerable massing of the epithelial cells occurs. As a rule rather more stroma is carried proportionately to the mass of tumor than in the case of the malignant adenoma.

In general the gross appearance of the adenocarcinoma is that of a shallow, ulcerated mass with irregular, raised, rather rough edges, and rather marked infiltration and induration of all the layers of the intestinal walls. Tendency toward annular growth is strongly marked, although extension occurs equally readily in the long axis of the gut.

The third group, the carcinoma simplex, corresponds to the more anaplastic tumors of Rankin and Broders' Groups III and IV. This group of carcinoma simplex is characterized by masses and clusters of close-packed, rather anaplastic epithelial cells without any evidence of alveolar formation, with frequent mitoses, (often abnormal), and marked variation in size of cells.

Also included in this group are the diffusely infiltrating signet-ring mucous secreting carcinomas, made up of isolated cells or very small clusters, similar to the diffusely infiltrating type seen in the stomach, which often gives rise to the Krukenberg tumor of the ovary.

The so-called colloid, or perhaps better, mucinous carcinoma, we have placed in a group by itself. This is essentially an adenocarcinoma, usually of rather anaplastic type. The characteristic feature is the presence of large amounts of mucus, not only within the alveoli, but more often lying between the tumor cells and their stroma. Thus the tumor cells are often widely separated from the supporting tissue and rarely may be difficult to find. This mucus is derived from the tumor cells, not from the stroma as is sometimes stated, and its extraglandular occurrence is explained on the basis of simple escape of secretion from within the glandular spaces or a disturbed polarity of the cancer cell, so that secretion takes place at its base.

Extension of the malignant process is facilitated by the wide separation of muscle fibers and connective tissue by mucus. Peristaltic contraction aids in the diffusion of the malignant cells. The strong tendency of the colloid carcinoma to peritoneal implantation metastases (30% as against 11% for adenocarcinoma) is also probably due to this manner of growth.

The gross appearance of colloid carcinoma is very striking, being a bulky mass, projecting but little into the lumen of the gut, constricting the lumen to a considerable degree, encircling early and penetrating diffusely through the wall. On section, even with the naked eye, strands of the muscularis can be seen still persisting, although widely separated, in the gelatinous secretion. Exophytic growth is relatively rare in this type of tumor.

In the case of the rectum we must consider two additional types of tumor, the epidermoid carcinoma, arising usually at the mucocutaneous junction, and extending upward into the rectum, undermining and later destroying the rectal mucosa, and the melanotic sarcoma, again occurring low in the rectum.

Curiously enough, in our group of sigmoid tumors we have one example of an unmistakable epidermoid carcinoma with definite keratinization occurring in a 55 year old female. This tumor was highly malignant, of very short duration, killing within six months of onset of symptoms. The patient appeared for treatment so late that nothing could be done. About the only explanation for the origin of this particular tumor is the occurrence of aberrant epidermal epithelium in the course of the sigmoid.

As will be seen from Table I, the great majority of intestinal cancers occur in the rectum and the adenocarcinoma is the commonest type. The high proportion of rectal tumors is probably due to the large number of patients with this lesion seeking treatment here. There is no significant difference in the numbers occurring in other portions of the large intestine.

TABLE I

DISTRIBUTION OF CASES BY SITE AND LESION

	Rectum	Sigmoid	Colon	Cecum
Malignant adenoma	21	3	4	3
Adenocarcinoma	52	12	16	13
Colloid carcinoma	9	3	1	7
Epidermoid carcinoma	4	1	—	—
Carcinoma simplex	2	—	—	1
Carcinoma, not stated	2	—	—	—
Melanotic sarcoma	1	—	—	—
Lymphosarcoma	—	—	—	1
Total	91	19	21	25

In a number of the adenocarcinomas and the malignant adenomas there is evidence that they originally arose from mucosal polyps. The polypoid origin of these carcinomas was quite definite in a number of instances, as evidenced by the occurrence of other polyps, which showed

II, III, IV and V. There is a close parallelism between the degree of malignancy of the tumor as determined on histologic grounds and the frequency of metastasis. Thus, in the rectum 67% of the malignant adenomas showed no evidence of metastases, whereas only 27% of the

TABLE II

CANCER OF THE RECTUM

	Total cases	M	F	Average age, years	Average duration before treatment	Not stated	Average duration after treatment	Total duration*	Not stated	Average duration untreated	No treatment or palliative treatment	Treated by operation	Treated by radium	Treated by x-ray	Combined treatment	With local recurrence	With no metastases
Malignant adenoma	21	12	9	62.1	1.5 yr.	4	6.5 mo.	1.6 yr.	—	2.1 yr.	12	6	3	—	—	2	14
Adenocarcinoma	52	20	32	61.5	1.5 yr.	12	10.0 mo.	1.8 yr.	2	1.2 yr.	20	18	8	3	3	8	14
Carcinoma simplex	2	1	1	55	4 mo.	—	5 mo.	9 mo.	—	1 yr.	1	1	—	—	—	—	—
Colloid carcinoma	9	5	4	57.2	1 yr.	1	10.2 mo.	1.3 yr.	1	8 mo.	4	4	—	1	—	1	5
Epidermoid carcinoma	4	—	4	65	1 yr.	1	1.8 mo.	2.5 yr.	—	—	1	2	1	—	—	1	3
Carcinoma, not stated	2	—	2	65	5 yr.	1	3 da.	2.8 yr.	—	—	2	—	—	—	—	—	—
Melanotic sarcoma	1	—	1	45	6 mo.	—	2 wk.	6 mo.	—	—	—	1	—	—	—	1	—

*Average total duration of treated and untreated cases.

as yet no malignant change, or at times by the structure of the growth, appearing in one part as a typical mucosal polyp, in another as cancer.

A considerable portion (38%) of the rectal group was operated upon, (Table II). Of these cases a moderate number died very soon postoperative, so the average duration of the cases after treatment is abnormally short. Again we must call attention to the fact that this series is not representative of the end results in operative cases but is rather heavily weighted by the occurrence of post-operative complications which one would naturally encounter in a hospitalized series of fatal cases. We have already pointed out[3] the high incidence of peritonitis, pulmonary embolus, hemorrhage and other postoperative fatalities in this group of cases.

As may be seen from Table II, some of the rectal cases were treated with radium. Naturally cases higher in the bowel were not so treated. The study of these radium-treated cases revealed the fact that although most of them had been apparently thoroughly treated, in no instance was the tumor entirely destroyed. This parallels our experience with surgical material previously treated by radium in which the continued growth of the tumor or the intense pain of radium burns has made operative interference imperative.

The condensed clinical information on the various cases grouped by the different parts of the large intestine involved, is shown in Tables

adenocarcinomas showed no metastases and both of the carcinoma simplexes had metastasized. The epidermoid carcinomas metastasized in only one instance, due perhaps to the fact that they were relatively heavily keratinizing, well-differentiated tumors. The colloid carcinoma is of particular interest in that of those occurring in the rectum, 56 per cent failed to show metastases, although from the clinical standpoint the colloid carcinoma is one of the highly malignant types.

The comparison of our data on age and duration with that presented by Karsner and Clark[4] is of interest. This is shown in Table VI, which is Karsner's Table IV, somewhat modified and amplified by our own cases. Our cases are older by several years in each group, although in most, the numbers are too few for real significance. The shorter duration of life in our cases and less definite parallelism between grades of malignancy and length of life are due to the character of our material, heavily weighted by post-operative deaths. We have considered our cases of malignant adenoma equivalent to their Grade I, our adenocarcinoma to their Grade II, and our carcinoma simplex together with colloid carcinoma as equivalent to their Grades III and IV.

The distribution of metastases in the carcinomas of the large intestine is chiefly by means of the lymphatic channels, although in some of the more highly anaplastic tumors, blood ves-

TABLE III

Cancer of the Sigmoid

	Total cases	M	F	Average age, years	Average duration before treatment	Not stated	Average duration after treatment	Total duration	Not stated	Average duration untreated	No treatment or palliative treatment	Irregular treatment	Treated by operation	Treated by radium	Treated by x-ray	Combined treatment	With local recurrence	With no metastases
Malignant* adenoma	3	1	1	58.3	5 mo.	—	5.1 yr.	5.3 yr.	1	—	2	—	1	—	—	—	1	2
Adeno-carcinoma	12	2	10	59.2	11.5 mo.	2	5.9 mo.	1.3 yr.	2	1 yr.	11	—	1	—	—	—	—	1
Colloid carcinoma	3	1	2	61.7	6 mo.	1	2.2 yr.	2.2 yr.	—	—	1	—	2	—	—	—	1	—
Epidermoid carcinoma	1	—	1	55	—	—	—	6 mo.	—	6 mo.	1	—	—	—	—	—	—	1

*1 case, sex not stated.

TABLE IV

Cancer of the Colon

	Total cases	M	F	Average age, years	Average duration before treatment	Not stated	Average duration after treatment	Total duration	Not stated	No treatment or palliative treatment	Irregular treatment	Treated by operation	Treated by radium	Treated by x-ray	Combined treatment	With local recurrence	With no metastases
Malignant adenoma	4	3	1	57.5	10.1 mo.	1	2 wk.	.9 yr.	—	2	—	2	—	—	—	—	4
Adeno-carcinoma	16	6	10	60.6	1.3 yr.	7	5.5 mo.	1.1 yr.	1	8	—	7	—	1	—	2	6
Colloid carcinoma	1	1	—	35	1 mo.	—	3 mo.	.5 yr.	—	1	—	—	—	—	—	—	—

TABLE V

Cancer of the Cecum

	Total cases	M	F	Average age, years	Average duration before treatment	Not stated	Average duration after treatment	Total duration	Not stated	Average duration untreated	No treatment or palliative treatment	Irregular treatment	Treated by operation	Treated by radium	Treated by x-ray	Combined treatment	With local recurrence	With no metastases
Malignant adenoma	3	1	2	65	10.5 mo.	—	1.8 wk.	.7 yr.	—	—	2	—	1	—	—	—	—	3
Adeno-carcinoma	13	7	6	58.8	5.4 mo.	4	9.4 mo.	1.0 yr.	1	6 mo.	8	—	3	—	2	—	—	4
Carcinoma simplex	1	—	1	65	—	—	—	.5 yr.	—	—	1	—	—	—	—	—	—	—
Colloid carcinoma	7	4	3	72.1	1.4 yr.	—	5.3 mo.	1.5 yr.	1	—	3	—	2	—	2	—	—	4
Lympho-sarcoma	1	1	—	65	—	—	—	—	1	—	1	—	—	—	—	—	—	—

sel invasion is noted. Of course in all but the lower portions of the rectum, vascular metastasis leads to the liver. In this connection it might be well to point out that nodules felt in the liver in the course of a laparotomy for carcinoma cannot be considered absolutely as metastases of the tumor. In at least two cases where definite nodules were felt by the surgeon and assumed to be carcinoma, subsequent postmortem examination revealed no metastases, the nodules in the liver being congenital cavernomas.

TABLE VI

AVERAGE AGE (YEARS) IN RELATION TO GRADES

	Grade I	II	III	IV
Rectum				
Stewart and Spies	58	55	55	47
Karsner and Clark	44	54	54	49
Warren	62.1	61.5	56.8	
Sigmoid				
Karsner and Clark	50	49	47	31*
Warren	58.3	39.2	61.7	
Upper colon				
Karsner and Clark	40*	45	54	40
Warren (including cecum)	60.7	59.8	67.2	

AVERAGE TOTAL DURATION (MONTHS) IN RELATION TO GRADES

Rectum				
Stewart and Spies	70	30	21	10
Warren†	19.2	21.6	13.8	
Sigmoid				
Warren†	63.6	15.6	26.4	
Colon				
Warren (including cecum)†	9.8	13.1	15	

*One case only.
†Autopsied cases only—time shortened by postoperative deaths.

In Table VII is shown the number of metastases of the different types of tumors of the rectum to the various organs. In contrast with this table is Table X showing the same data for the cecum that Table VII shows for the rectum, Table VIII for the sigmoid, and Table IX for the colon. These four tables are combined in Table XI.

The limited distribution of metastases as compared with cancer of the breast is very striking. There is no significant difference in the frequency of metastasis from tumors in different parts of the large intestine, but there is marked evidence that the power of metastasis and degree of malignancy tend to parallel one another. This is very clearly brought out in Tables XI and XII. A similar finding was noted by Rankin and Broders[1].

Of 31 malignant adenomas, 23 showed no metastases, and the metastases of the remaining eight involved only three sites: regional lymph nodes, liver, and lung. In contrast to this, 93 adenocarcinomas failed to metastasize in only 25 instances, and the secondary deposits were distributed among seven sites. The three cases of carcinoma simplex all metastasized. Of the 20 cases of colloid carcinoma 11 metastasized, involving seven different sites.

There were two cases of metastasis to bone, one of an adenocarcinoma of the rectum, the other of a colloid carcinoma of the rectum.

Probably had complete radiographic studies of the skeleton been possible in these cases, we would have found more instances of bone metastasis. Aufses[5] found eight metastases in 117 cases, while von Mielecki[6] reported three in 56 cases. Bone is one of the rarer sites of metastasis, however.

Aside from lymph nodes, the liver was most frequently involved (47 times) followed by the lungs (26 times).

The colloid or mucinous carcinoma is a particularly interesting type. While there may be considerable difference in the degree of anaplasia

TABLE VII

SITES OF METASTASIS OF CANCER OF THE RECTUM

	Regional nodes	Distant nodes	Adrenal	Bladder	Diaphragm	Heart	Large intestine	Liver	Lung	Muscle	Ovary	Peritoneum	Pleura	Skin	Spleen	Testis	Thyroid
Malignant adenoma	3	—	1	—	—	—	1	6	2	—	—	—	—	—	—	—	—
Adenocarcinoma	32	21	3	1	1	1	—	17	10	1	1	4	3	2	1	1	1
Carcinoma simplex	1	1	—	—	—	—	—	1	1	—	—	—	—	—	1	—	—
Colloid carcinoma	3	3	—	1	1	—	—	1	1	—	—	2	1	—	1	—	—
Epidermoid carcinoma	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Carcinoma, not stated	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Melanotic sarcoma	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Total	43	26	4	2	2	1	1	25	14	1	1	6	4	2	3	1	1

of the individual cells, nevertheless the secretion of mucus is so marked a feature that these tumors may be set apart as a distinct group. Very likely the group may be subdivided, but in view of the small number (20) in the present study it seems best to consider it as a unit. In all cases local extension and large bulk of the primary lesion were prominent features. Metastasis occurred in 11 of the 20 cases, involving 13 different sites, of which bone, heart and spleen were unusual. This tendency to wide metastasis, coupled with a bad ultimate prognosis, is our reason for placing this group in Grade III malignancy along with the carcinoma simplex.

Grading is of definite value in predicting the

TABLE VIII

SITES OF METASTASIS OF CANCER OF THE SIGMOID

	Regional nodes	Distant nodes	Adrenal	Diaphragm	Gall bladder	Heart	Kidney	Large intestine	Liver	Lung	Ovary	Peritoneum	Stomach
Malignant adenoma	—	—	—	—	—	—	1	—	1	—	—	—	—
Adenocarcinoma	9	9	1	1	—	—	—	—	7	4	1	2	—
Colloid carcinoma	1	1	1	—	1	1	—	1	2	1	—	1	1
Total	10	10	2	1	1	1	1	1	10	5	1	3	1

TABLE IX

SITES OF METASTASIS OF CANCER OF THE COLON

	Regional nodes	Distant nodes	Adrenal	Kidney	Liver	Lung	Pancreas	Peritoneum	Pleura
Adenocarcinoma	8	6	2	1	5	4	2	3	1
Colloid carcinoma	—	—	—	—	—	—	—	1	—
Total	8	6	2	1	5	4	2	4	1

TABLE X

SITES OF METASTASIS OF CANCER OF THE CECUM

	Regional nodes	Distant nodes	Diaphragm	Liver	Lung	Pancreas	Peritoneum	Pleura	Spleen
Adenocarcinoma	9	9	1	5	2	1	1	1	1
Carcinoma simplex	1	1	—	1	1	—	—	—	—
Colloid carcinoma	—	1	—	1	—	—	2	—	—
Lymphosarcoma	1	1	—	—	—	—	—	—	—
Total	11	12	1	7	3	1	3	1	1

TABLE XI

COMBINED LARGE BOWEL CANCERS

METASTASES TO IMPORTANT SITES

	Regional nodes	Distant nodes	Liver	Lung	Peritoneum
Malignant adenoma	3	—	7	2	—
Adenocarcinoma	58	45	34	20	10
Carcinoma simplex	2	2	2	2	—
Colloid carcinoma	4	5	4	2	6
Epidermoid carcinoma	1	—	—	—	—
Carcinoma, not stated	2	—	—	—	—
Melanotic sarcoma	1	1	—	—	—
Lymphosarcoma	1	1	—	—	—

TABLE XII

RELATION OF GRADE TO METASTASIS

	Number of cases	Number with metastasis	Without metastasis	
			No.	%
Malignant adenoma	31	8	23	74.2± 7.9
Adenocarcinoma	93	68	25	26.9± 4.6
Carcinoma simplex } Colloid carcinoma }	23	14	9	39.1±10.1

likelihood of metastasis, in contrast to the carcinomas of the breast[7]. Table XII shows a significant difference between the percentage of tumors failing to metastasize in the malignant adenoma and in the other types of tumors, but shows no significant difference between the two more malignant grades. The apparent tendency of the adenocarcinomas to metastasize more freely than the carcinoma simplex-colloid carcinoma group may be due in part to the small number of cases and in part to the inclusion in the latter group of nine cases of colloid carcinoma which died without evidence of metastasis.

Table VI also appears to indicate some connection between degree of malignancy and length of life, but obviously an autopsy series is of but little value in this regard.

SUMMARY

1. The distribution of metastases in 156 cases of cancer of the large intestine is reported.

2. The carcinomas may be divided into four grades on the basis of gross and histologic characteristics.

3. The colloid or mucinous carcinomas represent a varied group, probably between the adenocarcinoma and the carcinoma simplex in malignancy.

4. There is close parallelism between grade and extent of metastasis.

5. The extent of metastasis is relatively limited. The liver (47 instances) and lungs (26 instances) are most frequently involved, aside from lymph nodes.

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THE VOLUME AND HEMOGLOBIN CONTENT OF THE RED BLOOD CORPUSCLES IN THE LIGHT OF RECENT KNOWLEDGE OF ANEMIA*

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WHERE a simple and specific treatment exists for a particular disease, it is obviously important to diagnose the condition correctly. Recent laboratory procedures have greatly increased our ability to diagnose certain anemias for which we have at present very potent remedies. The determination of the average volume of the red blood corpuscles, and the average hemoglobin content and concentration, have been shown to be of value in giving information both as to the type of the anemia and the therapy which is indicated. The present report deals with some of these procedures and their clinical interpretation, and attempts to relate them to the modern concept of the etiology of certain anemias.

For excellent historical reviews of methods of determining the volume and hemoglobin content of red blood corpuscles the papers especially by Haden[1, 2, 3] should be consulted. These papers and those by Osgood[4, 5] and Wintrobe[6, 7] describe the results obtained by newer methods in normal subjects and in various diseases. The measurement of the diameter of the red blood corpuscles as demonstrated by Price-Jones[8] is closely related to this topic but is not for detailed comment here.

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METHODS

The methods of Wintrobe[6, 7] have been used in the present study and his nomenclature has been employed for the sake of simplicity of interpretation.

Exactly 5 cc. of venous blood is placed in a tube containing 0.05 cc. of a 20 per cent solution of potassium oxalate. After immediate and thorough mixing, a portion of this oxalated blood is placed in a Wintrobe hematocrit tube which is allowed to stand one hour, during which time a sedimentation test may be done. This tube is then centrifuged at high speed (2,500-3,000 revolutions per minute) and the per cent volume of cells read from the graduations on the tube. It is necessary for the centrifugation to be prolonged until the red blood corpuscles are completely packed, the exact length of time varying for different centrifuges. The per cent volume is multiplied by the factor 1.08 to correct for the shrinkage of the cells caused by the addition of the potassium oxalate. This is the corrected hematocrit per cent, or the per cent volume of the red blood corpuscles.

The number of the red blood corpuscles per cubic millimeter and the hemoglobin per cent are determined on the remaining oxalated blood. Pipettes and counting chambers certified by the U. S. Bureau of Standards are used. For the red blood corpuscle determination at least two chambers, usually four, are counted. The hemo-

globin is determined by the Sahli Haemometer, employing tubes, pipettes and standard which have been checked by the oxygen capacity method for determining the hemoglobin using the Van Slyke apparatus. Fifteen and six tenths Gm. of hemoglobin per 100 cc. of blood or an oxygen capacity of 21 volumes per cent, have been taken as the equivalent of 100 per cent hemoglobin.

The *mean corpuscular volume* (M.C.V.) is determined by dividing the hematocrit per cent ($\times$ 10) by the number of red blood corpuscles in millions per cu. mm., the result being expressed as cu. μ. For example, if the hematocrit per cent is 45 and the red blood corpuscles 5 million per cu. mm., the mean corpuscular volume is 90 cu. μ.

The *mean corpuscular hemoglobin* (M.C.H.) is determined by dividing the grams of hemoglobin per 100 cc. of blood ($\times$ 10) by the number of red blood corpuscles in millions per cu. mm., the result being expressed in micro-micrograms, symbolized by the letters $\gamma\gamma$. For example, if the hemoglobin per 100 cc. is 15 Gm., and the red blood corpuscles 5 million per cu. mm., the mean corpuscular hemoglobin is 30.0 $\gamma\gamma$.

The *mean corpuscular hemoglobin concentration* (M.C.H.C.) is determined by dividing the grams of hemoglobin per 100 cc. ($\times$ 100) by the hematocrit per cent, the result being expressed in per cent. For example, if the hemoglobin per 100 cc. is 15 Gm. and the hematocrit per cent 45, the mean corpuscular hemoglobin concentration is 33.3 per cent.

The mean corpuscular volume has the identical significance that the "individual cell volume" and the "volume index" have; the mean corpuscular hemoglobin the same that the "color index" has; the mean corpuscular hemoglobin concentration the same that the "saturation index" and the "hemoglobin index" of Whipple have. The terminology chosen here has the advantage, as stated by Wintrobe, that it expresses the actual size and hemoglobin content of the average red blood corpuscle in simple, logical terms. Moreover, the determinations do not require more or less chimerical normal figures which may vary for age, sex or geographical location.

MEAN CORPUSCULAR VALUES AND THEIR SIGNIFICANCE IN NORMAL SUBJECTS, PERNICIOUS ANEMIA AND HYPOCHROMIC ANEMIA

Table 1 shows the values for these determinations in 10 normal men, in a typical case of pernicious anemia and a case of hypochromic anemia ("secondary anemia") due to chronic blood loss from hemorrhoids. The color index is quite different in these three examples: in the normal men the color index is about 1.0; in the case of pernicious anemia it is about 1.5; in the case of hypochromic anemia it is about 0.45. Marked differences are also shown by the measurements of the volume and hemoglobin content of the corpuscles. The mean corpuscular volume and mean corpuscular hemoglobin in pernicious anemia in relapse are much greater than normal; in idiopathic hypochromic anemia and other types of hypochromic anemia they are much smaller than normal. The mean corpuscular hemoglobin concentration in pernicious anemia is approximately normal. It is a significant fact that in all blood disorders this factor never exceeds the normal. There is a certain maximum concentration of hemoglobin in red blood cells which cannot be exceeded, in other words a "saturation point". It is therefore somewhat incorrect to employ the term "hyperchromic" to some anemias. The concentration of hemoglobin in red blood corpuscles, however, is diminished in hypochromic anemia due to various causes. These points are further illustrated in Table 2.

The large red blood corpuscles which occur in the blood in pernicious anemia are verified by the measurement of the mean corpuscular volume. The recognition of the marked variation in the size and the shape of the red blood corpuscles, which is seen in a microscopic examination, is of great additional help in the diagnosis of pernicious anemia, and both the increase of size of the cells and the increased variation in size are brought out clearly by the painstaking methods of Price-Jones[8]. However, the measurement of the mean corpuscular volume together with the microscopic examination of a blood smear will usually be satisfactory in judging these characteristics. The observation of small red blood corpuscles which are seen under the microscope in the blood of hypochromic anemia may be checked by the measurement of the mean corpuscular volume. In pernicious anemia, in relapse, red blood corpuscles well filled with hemoglobin, and in hypochromic anemia red blood corpuscles poorly filled with hemoglobin (achromia), may be verified by measurement of the mean corpuscular hemoglobin and the mean corpuscular hemoglobin concentration. When an observer becomes ac-

TABLE 1

Types of cases	No. of cases	R.B.C.* mils per cu. mm.	Hgb. per cent	Hematocrit per cent	M.C.V. cu. μ	M.C.H. $\gamma\gamma$	M.C.H.C. per cent
Average determinations in 10 normal men		5.01	95.0	45.9	92	29.1	32.4
Pernicious anemia (one case)		2.02	59.5	28.9	141	45.9	32.7
Hypochromic anemia (one case)		3.37	29.5	19.5	58	13.6	23.6

*R. B. C. = Red blood corpuscles.
M. C. V. = Mean corpuscular volume.
M. C. H. = Mean corpuscular Hemoglobin.
M. C. H. C. = Mean corpuscular hemoglobin concentration.

TABLE 2

MEAN CORPUSCULAR VALUES IN VARIOUS CONDITIONS

(The extremes appear in light faced type; the averages appear in **BOLD** faced type)

Diagnosis	No. of cases	R.B.C.* mils per cu. mm.	Hgb. per cent	M.C.V. cu. μ	M.C.H. γγ	M.C.H.C. per cent
Normal men	10	4.6-5.7 **5.0**	89-100 **95**	85-97 **92**	28.2-32.5 **29.8**	30.0-34.5 **32.4**
Normal women	13	4.1-5.0 **4.6**	80-92 **84**	87-98 **93**	26.3-31.3 **28.5**	29.1-34.6 **31.0**
Idiopathic hypochromic anemia with achlorhydria	24	1.9-5.0 **4.0**	16-74 **47**	53-87 **70**	11.4-26.6 **17.9**	18.9-31.8 **25.2**
Anemia due to chronic blood loss	23	1.2-4.6 **3.0**	8-59 **40**	58-88 **77**	10.3-24.5 **19.3**	13.5-29.9 **24.8**
Anemia associated with defective diet	8	3.7-4.8 **4.2**	52-82 **71**	79-97 **87**	21.3-30.8 **25.7**	26.7-31.7 **29.4**
Anemia in pregnancy	27	1.4-4.8 **3.4**	35-80 **48**	61-115 **80**	14.9-37.9 **22.8**	23.2-33.1 **28.2**
Pernicious anemia	37	1.0-4.7 **2.9**	21-91 **62**	93-153 **116**	28.5-45.9 **35.5**	27.3-34.4 **30.6**
Anemia due to acute blood loss	10	1.0-3.5 **2.0**	19-53 **35**	91-126 **107**	21.2-33.9 **28.4**	22.1-29.9 **26.5**
Hemolytic jaundice (various types)	4	1.0-3.8 **2.7**	30-80 **58**	89-164 **118**	30.7-45.8 **35.6**	28.0-34.7 **31.9**
Acute cardiac decompensation	3	3.9-6.1 **5.2**	75-110 **97**	95-109 **102**	28.3-29.7 **29.0**	27.3-29.8 **28.6**
Polycythemia rubra vera	5	7.7-9.2 **8.2**	110-163 **135**	66-93 **80**	21.8-30.3 **25.8**	30.0-34.7 **32.4**
Macrocytic anemia of obscure origin (including aplastic anemia)	6	0.9-3.0 **2.0**	20-62 **43**	102-138 **114**	26.8-40.8 **33.4**	28.3-31.2 **29.3**
Benzol poisoning	1	1.2	15	128	37.9	29.9
Chronic myelogenous leukemia	8	1.5-4.9 **3.0**	33-71 **52**	81-103 **90**	21.8-33.0 **28.2**	29.3-34.0 **31.1**
Chronic lymphatic leukemia	1	4.1	72	88	27.7	31.3
Osteosclerotic anemia	1	2.2	37	107	26.0	24.2
Chronic nephritis	23	1.6-4.5 **3.1**	26-94 **58**	81-109 **96**	22.9-36.6 **29.7**	23.4-33.8 **31.1**
Pellagra	5	3.3-4.5 **3.9**	67-86 **76**	95-112 **103**	28.4-33.4 **31.0**	28.6-32.2 **30.3**
Cirrhosis of the liver	2	1.9-2.9 **2.4**	38-48 **43**	96-115 **105**	25.6-31.0 **28.3**	26.5-27.0 **26.7**
Lead poisoning	4	3.1-3.4 **3.3**	60-66 **63**	88-96 **92**	26.8-30.2 **28.0**	28.0-33.6 **30.4**
Cooley's anemia (erythroblastic anemia)	1	3.9	51	69	20.4	29.4
Idiopathic purpura hemorrhagica	1	4.1	65	84	24.8	29.5
Hemophilia (without recent blood loss)	2	4.8-5.1 **5.0**	78-84 **81**	83-85 **84**	23.9-27.2 **25.6**	28.7-31.8 **30.3**
Catarrhal jaundice	1	4.3	91	112	33.2	29.6

*R. B. C. = Red blood corpuscles.
M. C. V. = Mean corpuscular volume.
M. C. H. = Mean corpuscular Hemoglobin.
M. C. H. C. = Mean corpuscular hemoglobin concentration.

customed to the use of a given degree of magnification, he can, in the study of pronounced anemia, tell with ease the approximate size and hemoglobin content of red blood corpuscles. In general, the microscopic examination of the blood smear remains the best single laboratory method of diagnosing the type of anemia, and many impressions obtained from scrutiny of a blood smear may be verified by these new and accurate methods.

In recent years, particularly since the discovery by Minot and Murphy of the successful method of treating pernicious anemia by means of liver, there has been a considerable growth of knowledge concerning the nature of certain types of anemia. Dietary deficiencies are coming to be regarded as of primary importance in the production of anemia. The important concept of "conditioned deficiencies" has been elaborated by Castle and his co-workers[9]. A patient with pernicious anemia may not have any insufficiency of his diet necessarily, but he has a lack of a specific substance in his stomach secretion which prevents the adequate formation of necessary blood-building substances from the diet. Anemia associated with chronic diarrhea, in which a defect in the absorptive mechanism of the gastro-intestinal tract prevents the entrance into the body of blood-building material, is also an example of a "conditioned deficiency". Idiopathic hypochromic anemia, occurring typically in middle-aged women with reduction and usually absence of free hydrochloric acid in the stomach, is probably a "conditioned deficiency". Here, iron is the most important deficient substance[10] which is prevented, perhaps, by a gastro-intestinal abnormality, from being utilized in a normal manner to form new hemoglobin. The remedy for such types of anemia is of course to give an adequate (large) amount of the deficient substance to the patient.

As a working hypothesis to explain the striking differences in the mean corpuscular values between pernicious anemia and hypochromic anemia, it may be assumed that the manufacture of stroma and hemoglobin are dependent upon quite separate mechanisms. In pernicious anemia it may be assumed that there is a deficiency of stroma-building substance, that cell maturation is inhibited, although hemoglobin formation takes place in a normal fashion, and therefore the few cells which are being manufactured are saturated with hemoglobin and are in most instances large in order to carry as much hemoglobin as possible. In hypochromic anemia it may be assumed that it is particularly hemoglobin-building substances that are deficient, and that therefore a normal number of cells may be manufactured which carry only a small quota of hemoglobin and are therefore small. Such an hypothesis is useful and is borne out by observations upon other states of anemia, as will be described. It is very diagrammatic, however, since undoubtedly stroma and hemoglobin formation are very intimately linked.

In uncomplicated pernicious anemia, liver extract, or similar potent material, is alone effective in alleviating the anemia. In hypochromic anemia, due to one or several factors known to produce an iron deficiency (diet restricted in iron-containing foods, blood loss, gastric achlorhydria, etc.), iron therapy alone is effective. In the first instance, liver extract may be regarded as a stroma-building substance; in the second, iron may be regarded as a hemoglobin-building substance. Very commonly, however, anemia is due to several different causes and the characteristics of the blood are altered according to the predominant cause; for example, in certain cases of pernicious anemia having an accompanying iron deficiency[11], in sprue[12], and in pregnancy anemia[13] the deficiency of stroma-building substance or the deficiency of hemoglobin-building substance may at different times predominate. In these mixed conditions, mean corpuscular volume and hemoglobin determinations will be of great help in indicating the type or types of therapy which should be employed.

When suitable therapy is employed the blood improves, and at the same time the volume and hemoglobin content of the red blood corpuscles return gradually to normal. Figures 1, 2 and 3 represent the blood findings in three different cases of anemia during the course of recovery. The first, a case of pernicious anemia, had the usual response to the administration of a potent substance (liver extract). The mean corpuscular volume, and the mean corpuscular hemoglobin, which were quite typical of pernicious anemia fell during recovery nearly to normal. The mean corpuscular hemoglobin concentration remained, for the most part, in the normal range.

The second case (figure 2), a case of idiopathic hypopathic anemia with achlorhydria, showed a gradual increase of the mean corpuscular volume and hemoglobin content toward normal as the hemoglobin increased under iron therapy.

The third case (figure 3) was one of chronic blood loss due to peptic ulcer with a recent acute hemorrhage. When the patient was first observed there was a reticulocyte response of 18 per cent to acute blood loss, and a mean corpuscular volume at this time of 109 cu. μ. Such an increase of the mean corpuscular volume is a common finding in acute blood loss when the number of reticulocytes is increased. The hemoglobin-building reserve was low, as shown by the inability to gain hemoglobin in spite of an increase in red blood corpuscles. The new red blood corpuscles were very poorly

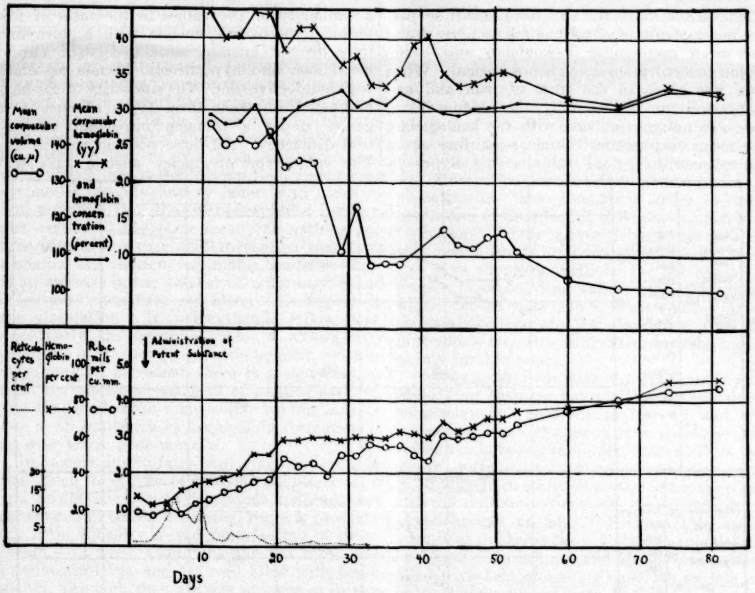

FIG. 1. The red blood corpuscles, hemoglobin, and mean corpuscular values in a case of pernicious anemia receiving treatment with potent substance. (100 per cent hemoglobin = 15.6 Gm. per 100 cc.)

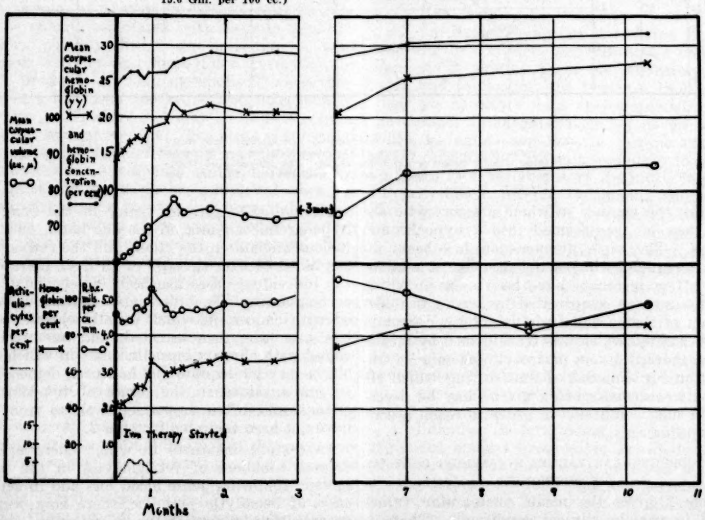

FIG. 2. The red blood corpuscles, hemoglobin, and mean corpuscular values in a case of idiopathic hypochromic anemia with achlorhydria receiving treatment with iron. (100 per cent hemoglobin = 15.6 Gm. per 100 cc.)

filled with hemoglobin and were small, so that the mean corpuscular volume fell to normal and the mean corpuscular hemoglobin and hemoglobin concentration much below normal. When iron was given in the form of iron and ammonium citrate, 6 Gm. daily, these latter values rose gradually to normal with the hemoglobin, the mean corpuscular volume remaining at an approximately normal value. This figure illustrates the manner in which a macrocytic anemia may be transformed into a hypochromic anemia. The same phenomenon has been observed frequently in pernicious anemia, and indeed a few instances have been seen in which this disease was complicated by severe iron deficiency so that a blood picture of hypochromic anemia existed which was transformed by means of iron therapy into a macrocytic anemia[11]. Observation for a period of time during which effective treatment is being given, may be necessary in such conditions in order to reach a conclusive diagnosis.

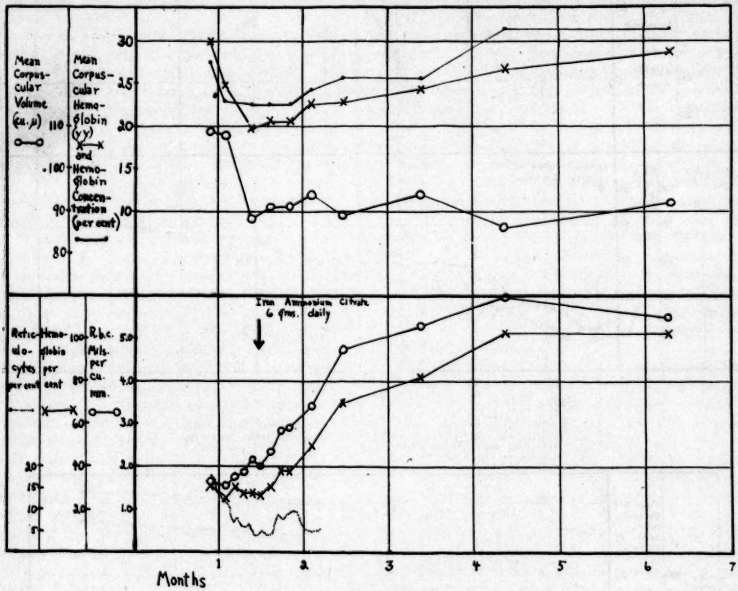
Mean Corpuscular Volume (cu. μ)
Mean Corpuscular Hemoglobin (yy) and Hemoglobin Concentration (per cent)
Iron Ammonium Citrate 6 gms. daily
Reticulocytes per cent
Hemoglobin per cent
R.b.c. Mils. per cu. mm.
Months

FIG. 3. The red blood corpuscles, hemoglobin, and mean corpuscular values in a case of chronic blood loss due to peptic ulcer with a recent acute hemorrhage. The influence of treatment with iron. (100 per cent hemoglobin = 15.6 Gm. per 100 cc.)

MEAN CORPUSCULAR VALUES IN VARIOUS CLINICAL CONDITIONS

Table 2 gives the mean corpuscular values found in various clinical conditions. There is a similarity of the values in the cases of idiopathic hypochromic anemia with achlorhydria, hypochromic anemia associated with chronic blood loss, and hypochromic anemia associated with a defective diet. The similarity of the blood findings links these three types of anemia together under a common important etiology: iron deficiency. All three respond well to iron. The values for pregnancy anemia have wide variation, the extremes being in the range of hypochromic anemia on the one hand, and pernicious anemia on the other, and the response of the blood to iron therapy or to liver therapy in the individual cases has been closely related to corresponding blood findings. Anemia associated with cancer, in which usually chronic blood loss had been demonstrated, and anemia associated with chronic sepsis in cases in which usually a very inadequate diet had been demonstrated, gave values in the range of hypochromic anemia and often responded well to iron, but have not been included in table 2.

In certain instances in which there are increased numbers of reticulocytes in the blood stream, following acute blood loss and in certain cases of hemolytic jaundice, there may occur a greater than normal mean corpuscular volume

and mean corpuscular hemoglobin. Cases of hemolytic jaundice with normal or reduced mean corpuscular volume have been reported. However, the greatest value for mean corpuscular volume obtained in the present series of cases was 164 cu. μ, in a case of hemolytic jaundice having a pronounced crisis in which the reticulocytes had reached a level of 60 per cent. Reticulocytes are usually larger cells than non-reticulated adult cells[14], and it would seem that these young corpuscles, usually heavily laden with hemoglobin, are supplying the emergency need for oxygen carriers. The large mean corpuscular volume in acute cardiac decompensation may have a similar explanation, for in this condition there is an increase in the number of reticulocytes and often very immature red blood cells appear in the circulation. In polycythemia rubra vera, apparently the opposite state of affairs may exist, for in this condition, although the reticulocytes may be increased, there is presumably no need for increased numbers of oxygen carriers, and the small mean corpuscular volume may be due to an inhibition of hemoglobin formation in an over-active bone marrow.

In anemia due to fatty bone marrow degeneration, such as occurs in idiopathic aplastic anemia, or aplastic anemia dependent upon a known cause such as benzol poisoning, there is presumably little difficulty in the manufacture of hemoglobin. In these conditions the red blood corpuscles are large and are well filled with hemoglobin, although they are not so large as in pernicious anemia in pronounced relapse. The same occasionally occurs in myelophthisic anemia due to leukemia, or present in osteosclerotic anemia.

The anemia associated with chronic nephritis is very possibly due to an inhibition of bone marrow activity and hemoglobin formation by toxic products of body metabolism, whose normal exit has been blocked. Not infrequently, however, a defective diet may be in part responsible for such an anemia. The mean corpuscular volume in this type of anemia may be increased or decreased. A response of the blood to iron has on several occasions been observed when the mean corpuscular volume and hemoglobin were reduced, and when there was a definite history of a prolonged defective diet. In general, however, therapy has been of little benefit in anemia associated with chronic nephritis.

In five cases of pellagra slight anemia was found, and in four of these the mean corpuscular volume was increased. In anemia associated with cirrhosis of the liver the mean corpuscular volume of one out of two cases was increased, whereas the hemoglobin concentration was reduced in both. In four cases of lead poisoning with moderate anemia the mean corpuscular volume was within normal limits, but the mean corpuscular hemoglobin concentration in three was diminished. The values in a case of Cooley's anemia (erythroblastic anemia), a case of idiopathic thrombopenic purpura hemorrhagica, two cases of hemophilia, one case of catarrhal jaundice, and two cases of myxedema are given in table 2.

COMMENT

The changes in the volume and hemoglobin content of the red blood corpuscles have been described in various diseases, particularly in pernicious anemia and in hypochromic anemia. An attempt has been made to give a logical explanation for these changes in order to clarify the nature of the effect of the diseases upon blood formation. These newer laboratory tests not only give a better understanding of anemias but also give simple indications for the use of the proper blood-building substances. The anemia which accompanies some diseases, such as cirrhosis of the liver and lead poisoning, gives findings which are often difficult to explain, and requires further study.

New criteria, both clinical and laboratory, for classifying anemias, have caused a confusion in nomenclature. New names for old and well-recognized types of anemia have been given and old names have lost their significance. Thus "secondary anemia" can no longer mean exclusively an anemia in which the color index is reduced and the red blood corpuscles are small and pale (hypochromic anemia). For example, in the anemia of nephritis the red blood corpuscles may be larger than normal and on the average well filled with hemoglobin. Pernicious anemia may be no longer placed with the group "primary anemia", and to call pernicious anemia "secondary anemia" would lead to misunderstanding.

At the present stage of knowledge it seems practical to consider the clinical, or etiologic, diagnosis separately from the laboratory diagnosis, and to combine the two in a simple form when the necessary data are completed. Thus, the clinical (etiologic) diagnosis of an anemia which is found sometimes in chronic nephritis would properly be "anemia secondary to chronic nephritis". The laboratory diagnosis might be "hypocythemic, macrocytic anemia, and normochromic anemia". The completed diagnosis would then be "macrocytic anemia secondary to chronic nephritis". A thorough discussion of this subject is not within the scope of this paper. It is necessary to state, however, that the frequently used term "hypochromic anemia" applies to a subdivision of the old classification "secondary anemia", in which the red blood corpuscles tend to be small (microcytic) and poorly filled with hemoglobin, and it applies particularly to those anemias which respond well to iron.

A difficulty in terminology, however, is no argument against progress in knowledge. The procedures which have been described represent a considerable advance and are certain to be very useful in the field of general clinical medicine.

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PERINEPHRIC ABSCESS*

BY CHANNING S. SWAN, M.D.†

IN Volume XXI, pages 1-5, of *Surgery, Gynecology, and Obstetrics*, for July, 1915, Dr. Edward P. Richardson reviewed the fifty-nine cases of perinephric abscess that were found in the records of the Massachusetts General Hospital in the years 1899 to 1913. This present paper reviews the one hundred and fourteen additional cases that have occurred from 1913 to 1932. We will consider factors bearing on the etiology, the clinical picture, and the treatment of this condition.

ETIOLOGY

Dr. Richardson's paper stresses and gives an excellent discussion of the question of etiology of perinephric abscesses, the diagnosis of which is at times quite obscure. The classification outlined in that paper needs no change at the present state of our knowledge. Graphically this classification is as follows:

(1) Origin in perirenal fat.
(2) Extension from organs,
  (a) Kidney
  (b) Other organs such as pleura, ribs, spine, appendix, colon, etc. These last cases are merely geographic perinephric abscesses and could rightly be called either lumbar or retroperitoneal abscesses. Also at times it is difficult properly to classify an abscess as to whether it is sub-diaphragmatic or perinephric.

The other way of grouping is

(1) Primary (including traumatic).
(2) Secondary.

The primary group includes those that have arisen through the blood stream or lymphatic distribution from outlying septic foci. Whether the blood stream source comes direct to the perirenal fat or indirectly by first causing a lesion of the renal cortex or fibrous capsule is still in doubt. There appears to be clinical and experimental evidence to support both ideas. The hematogenous kidney is well recognized as a clinical entity. This fact supports the view that the abscess results by extension from a renal blood-borne origin, even if there is but a very slight renal involvement. Cortical abscesses undoubtedly give rise to perinephric abscesses. The urine is usually or often negative in such cases. However, in operating for perinephric abscess one seldom explores the kidney sufficiently to determine definitely the actual focus for the septic process, whether it arose from a cortical abscess or by direct metastases to the perirenal fat. Probably there is an actual renal start for the majority of the primary metastatic cases; for why should the perirenal fat have such a predilection for picking up metastatic septic processes? The lymphatic channel is important in ascending and some descending infections into the perirenal area.

Histories of cases typifying the divisions of classification given above are as follows:

(1) Primary metastatic case.

M. G. H. Case No. 319950. Male—Aged 33. Patient entered the hospital March 13, 1932, complaining of pain in the right fourth finger for four days. He had injured it on a nail eight days previously while at work on a box he was opening. Incision and drainage of the tendon sheath was done on the day of entrance. A culture showed staphylococcus aureus. On March 30, two and a half weeks later, patient developed pain in right lumbar region and had a continued elevation of temperature. White count was 14,000. Urine was constantly negative, and x-ray showed the diaphragm high with limited motion on the right side. On April 8 I drained a perinephric abscess in the right lumbar region. There was a large cavity, admitting four fingers, which was filled with thick yellow-white pus. A culture of this showed staphylococcus aureus. The convalescence from then on was uneventful. The kidney was not explored in this case, but this is a typical story of what is considered a primary blood-borne metastatic perinephric abscess from an outlying focus of infection.

*Read at a clinical meeting of the Staff of the Massachusetts General Hospital, December 8, 1932.

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(2) Two primary traumatic cases.

M. G. H. Case No. E. S.-216840. Male—Aged 10. Patient entered the hospital August 18, 1917. Three weeks before he had been struck by another boy in the left lower back, since when he had had considerable pain, especially on walking. For the past two weeks he had been confined to bed for this reason. There were no urinary symptoms. Examination revealed a swelling the size of a small orange, not reddened or tender, in the left back over the kidney region. An x-ray was negative. The urine was negative. The white count was 17,000. On August 20, 1917, incision and drainage for perinephric abscess was performed by Dr. W. A. Perkins. The abscess was found behind and above the kidney and consisted of a large cavity containing several ounces of thick yellow pus. The cavity ran up as high as the diaphragm. The bacteriological report from the culture showed no growth. Following the operation the child made an uneventful convalescence, and was discharged from the hospital September 9, 1917.

M. G. H. Case No. E. S.-204351. Male—Aged 9. Patient entered the hospital September 21, 1915. Two weeks previously the patient fell from a bridge into a river striking the left side of his back on a rock. He has since complained of severe pain over the region of the left kidney. For the last few days this has been much worse and he has had chills and fever. Examination showed a swollen, tender, fluctuant area the size of the palm of a hand on the left side of the back just under the twelfth rib and extending around toward the front. Temperature was 101.5°. Urine was negative for pus. Operation was performed the same day by Dr. F. G. Balch. A three-inch vertical incision was made just below the costovertebral angle and four inches to the left of the spine. This was carried through the lumbar muscles to the perinephric space where two ounces of thick yellow pus were evacuated. The patient made an uneventful convalescence and was discharged from the hospital on the eleventh day postoperative.

These are typical stories of a sterile perinephric abscess developing in the perirenal fat, undoubtedly from breaking down of a hematoma.

(3) Primary cases of probable lymphatic origin.

Richardson reported a case of perinephric abscess which arose following the divulsion of a stricture of the urethra and which he claimed showed the possibility of infection from the lower urinary tract by way of the lymphatics. M. G. H. Case No. 303837 quoted below as an example of a perinephric abscess resulting from original pathology above the diaphragm is also of very probable lymphatic source. Richardson states, "the communication of the lymph-vessels of the cortex with those of the perinephric fat through the fibrous capsule; of the ureter with those of the kidney and bladder; the connection of the lymphatics of the perinephric fat with those of the diaphragm; the further connections through the lumbar, iliac, and hypogastric nodes suggest many possible sources of infection through the lymphatics."

(4) A case secondary to and dependent upon extension from well-established renal disease.

M. G. H. Case No. 300994. Male—Aged 56. Entered the hospital August 20, 1929. Two months previously he had had his appendix taken out because of an attack of pain in the right side and right lower quadrant. (We were unable to determine whether the appendix was pathological.) Following operation the patient had had to be catheterized. Several days later he developed pain in the right flank. (We do not know whether the patient was infected by catheterization or whether his whole illness began with pyelitis.) He had definite urinary symptoms with frequency eight to ten times during the day, and twice at night. Temperature rose to 102° daily. White count was 17,000 and the urine was loaded with pus. He did not, however, appear to be extremely ill. Cystoscopy revealed pus coming from the right kidney, and a pyelogram on that side showed slight hydronephrosis and blunting of the calices. There was no suggestion made by the x-ray of any possibility of a perinephric abscess. Following the ureteral catheterization and lavage of the renal pelvis, the temperature dropped for several days; then, however, it again arose and was unrelieved by cystoscopic intervention. There was no mass in the flank but because of the failure to relieve him by cystoscopic measures I did an exploration on September 4, 1929, and found a perinephric abscess containing a large cavity with much pus in it. The abscess was just below and lateral to the kidney. Culture showed staphylococcus aureus. Following this drainage the patient's condition improved for a while, but again there was elevation of temperature unrelieved by further cystoscopic manipulation. The patient's condition was becoming rapidly worse, therefore a transfusion of blood and a nephrectomy were performed on October 11, 1929, by Dr. J. D. Barney. A right kidney totally destroyed by inflammation was removed. The patient's postoperative course was stormy, and he died several days later. Postmortem examination revealed staphylococcus aureus septicemia and multiple abscesses in the lungs and left kidney.

This case has many lessons. In the first place, the patient may never have had appendicitis. The whole illness may have been a genitourinary infection from the beginning. If it was not, then there is the likelihood that following the removal of a pathological appendix he had become infected by careless catheterization. The events that developed subsequently show how serious infection introduced in this manner may sometimes be. Furthermore, this case brings up the question as to when it is wise to stop conservative treatment of the infected kidney by the cystoscope and resort to operative interference. Experience with several virulent renal infections with the patient not appearing so very ill during the conservative treatment has led me to advocate surgical interference in some cases of renal infection that might have been cleared up with less radical measures. However, it is cases like the one just quoted that cause me to lean to the more radical procedure for safety's sake whenever there arises any question as to the efficacy of conservative therapy. This surgical interference in the absence of finding a perinephric abscess consists of draining the kid-

ney through a nephrotomy incision. In this case it was done too late.

(5) A case arising from extension from an organ other than the kidney, i.e., the pleura.

M. G. H. Case No. 303837. Male—Aged 27. Entered the hospital January 3, 1930, complaining of pain in the back of five weeks' duration. Patient stated that he had carried a girl in one arm up a flight of stairs. Two days later he had felt a dull pain in his left back under the lower ribs. The pain was especially severe on deep or quick breathing. There was also a slight unproductive cough. His temperature had risen to 102° and a local doctor had found signs of fluid in the left pleural cavity. Three weeks later his temperature was running up to 104° in the afternoon. The pain on breathing had stopped and there were no longer signs of fluid in the pleural cavity. He had, however, definite left costovertebral tenderness and he had lost forty pounds in weight. Examination when he entered the hospital revealed a localized tender swelling in the left costovertebral angle. The urine was negative. The white count was 21,000. On January 6, 1930, I explored him through a usual left lumbar incision and ran into a large abscess cavity just under the soft parts. This abscess ran high and extended from the diaphragm to the region behind the kidney. Convalescence was uneventful following the evacuation of the pus. The culture showed staphylococcus aureus.

This case is given as an example of a perinephric abscess that arose from extension of an inflammatory process to the area surrounding the kidney but had nothing to do with the kidney itself. Extension was probably made through the lymph channels, there being a definite connection between the lymphatics of the diaphragm and those of the perinephric fat. Such cases, I feel, are equally properly classified as subdiaphragmatic, lumbar, or retroperitoneal abscesses, depending upon their site of origin.

The etiology from the bacteriological point of view as shown from the cultures taken at time of operation consistently shows a preponderance of instances of staphylococcus aureus infections, thus confirming the findings of others who have reviewed similar series of cases. Cultures apparently were not taken in every case but results are recorded in fifty-three cases. They are as follows:

Staphylococcus aureus	30 cases
Staphylococcus	9 cases
Streptococcus	6 cases
Colon bacillus	5 cases
Tubercle bacillus	2 cases
Streptococcus and Staphylococcus (mixed)	1 case

The clinical etiological factor was unknown in the vast majority of the cases. However, the following statistics are available expressed in the number of cases in which each occurred: boils eleven, trauma nine, renal disease seven, tuberculosis six, upper respiratory infection five, carbuncle three, abscess of arm two, septic hand one, osteomyelitis of spine one, prostatic abscess one, mastoiditis one, empyema two. The seven cases with renal pathology other than tuberculosis that gave rise to perinephric abscesses included the following pathology: calculus associated with advanced renal infection, four cases; renal neoplasm with pyelonephritis, one case; renal abscess from long-standing urinary infection, two cases.

The male sex was much more frequently affected than the female. There were 102 male cases and 12 female cases. The cause for this is not at all clear.

Whether the abscess will occur on the right or left side appears to be about an even chance. In this series 60 cases involved the left side, 50 cases the right side, one case was bilateral, and in three cases no mention was made of which side was affected.

The age of the patient had some bearing on the etiology. In general, the abscesses that were metastatic occurred in young adults, whereas those that were the direct result of a long-standing renal infection occurred more often in people of middle age. Going into the matter of age in a little more detail one finds that, divided by decades, the cases occurred as follows:

Up to ten years old	6 cases
Between ten and twenty years old	21 cases
In their twenties	32 cases
In their thirties	33 cases
In their forties	13 cases
And over fifty	9 cases

As evidenced by these statistics perinephric abscess is predominately a disease of young adult life.

Thus far we have stressed various factors having a bearing on the etiology of perinephric abscess. We will now consider in detail the factors which contribute to the clinical picture of this condition.

CLINICAL PICTURE

It is a frequent and truthful saying that the diagnosis of a perinephric abscess is often very obscure and difficult. The mistaken diagnoses with which the cases were sent into the hospital are legion, as enumerated below. Some cases had been operated on for other seeming pathology. However, despite these facts it is also true that in the vast majority of the cases in this series a correct diagnosis was made fairly promptly before or soon after admission to the hospital. The majority of the histories are quite typical and the symptoms classical and localized. Most errors are made in those cases that present generalized, slowly developing, indefinite symptoms. A list of the mistaken diagnoses in these cases includes empyema, typhoid fever, psoas abscess, appendix abscess, neoplasm of ilium, echinococcus disease, amoebic disease of the liver, septic hip, abscess of groin, malignant disease of the colon, pneumonia, grippe, neuritis,

intestinal obstruction, salpingitis, perihepatitis, cholecystitis, abscess of back, necrosis of rib, hematogenous kidney, kidney abscess, renal neoplasm, tuberculous spine, lymphoma, retroperitoneal abscess, subphrenic abscess, osteomyelitis, pyelitis, and renal calculus. Thus we have mistaken diagnoses that range from being nearly correct or at least associated with the correct diagnosis, to those that are a far cry from the actual condition. It is interesting to note that although a normal appendix was removed by mistake in two cases, in two other cases a perinephric abscess developed in the hospital after removal of a definitely pathological appendix.

In the metastatic cases the length of time elapsing between the onset of the original outlying focus and the development of signs of a perinephric abscess varied considerably but was always a matter of a few to several weeks. The duration of the patient's symptoms due to a developing perinephric abscess before surgical attention was given also varied a good deal, depending on how clear-cut the picture was. One to three or four weeks was common for the "present illness", and a history dating back two months was not uncommon. Indeed there were some cases that dated back several months, probably representing incidences of long-standing, non-suppurating perinephritis. Once the patient got to the hospital, operation was performed fairly promptly. Fifty-seven of the cases were operated on within one to four days. In other cases the diagnosis was sufficiently obscure and the patient not so ill but that a thorough investigation was felt to be warranted to establish a definite diagnosis. A few cases actually developed in the hospital while the patient was recovering from another illness such as a septic hand, carbuncle, or acute appendicitis.

Considering the onset and subsequent chain of symptoms, it may be said that most of the cases started with acute pain in the flank or back, fever and tenderness. In eighty-three cases there was definite pain in the kidney region. Other cases had pain in the lower quadrant or in the hip, and occasionally the pain was localized in the lower part of the chest. The cases with indefinite generalized symptoms would begin possibly with chills, fever, malaise, weakness, loss of appetite and some gastro-intestinal symptoms such as nausea, vomiting, gas, and diarrhea. Then, later on, say one to three weeks hence, they would begin to get localizing signs such as pain, swelling, and tenderness in a kidney region.

Definite urinary symptoms occurred in less than one-third of the cases. The chief urinary symptom was frequency; some cases also reported dysuria or that the urine was "high colored". The urinalysis was recorded in 110 cases. In only 25 cases was the urine loaded microscopically with pus. In 50 cases there were rare, occasional, or a few pus cells. In 35 cases there was no pus whatsoever found in the urine. Some cases would show pus at one analysis and not at another. Apparently the majority of cases do show some pus in the urine at some time or other. Many cases had pus in the urine without any urinary symptoms. Very little that was helpful was obtained from urine cultures. On the whole, urinary symptoms, urinalysis and cultures are not constant or dependable factors in the diagnosis of perinephric abscess.

The white count varied from 11,000 to 40,000. By far the commonest readings were between 18,000 and 22,000. Leucocytosis is practically a constant factor.

Cystoscopy was performed in twenty-nine cases. Although undoubtedly a warranted procedure in the majority of cases, still it is quite likely to give only negative evidence. Eight of the twenty-nine cystoscopies were negative. Pus was revealed coming from the kidney on the affected side in fourteen cases. In two cases a pyelogram revealed a renal pelvis deformed by pressure from an extrinsic mass. This, of course, was very helpful. Four cases revealed a double ureter and one a hydronephrosis on the affected side. The cystoscopies were done mostly on those cases that had pus in the urine and definite urinary symptoms.

The x-ray was of no diagnostic help in the vast majority of the cases. However, it is helpful in enough cases to warrant its routine use. The two typical x-ray signs as first mentioned by Lipsett and popularized by Beer are (1) the curving of the spine away from the involved side, approximating the iliac crest and last ribs on the diseased side, and (2) the obscuration of the outer margin of the psoas muscle. In only nine cases was a definite diagnosis of perinephric abscess made by x-ray in this series. However, not infrequently the x-ray showed a high diaphragm with limited motion on the affected side. With other clinical evidence of perinephric abscess an x-ray report as just mentioned should considerably strengthen one's conviction of the right diagnosis. "Large kidney", "area of increased dullness in kidney region", "abscess of kidney", are some of the x-ray diagnoses that were close enough to be helpful. However, on most of the cases no x-ray evidence is obtained.

The advanced cases with chills, loss of weight and septic mien reveal how a perinephric abscess can spread. Extension may occur upwards sufficiently to give a raised diaphragm, backward and laterally enough to give a tumor mass in the lumbar region evident to the eye, downward on the psoas muscle behind the peritoneum to a point in the groin above Poupart's ligament, or extending below Poupart's liga-

ment to make itself evident in the femoral triangle or the region near the hip joint; forward extension has occurred to cause a liver abscess or a peritonitis, and in these extreme cases extension into the blood stream has caused fatal septicemia.

TREATMENT

Most cases are relieved by simple incision and drainage of the abscess cavity. At times, however, secondary operations have to be performed, not only for the removal of a destroyed kidney, but also further drainage operations for unopened cavities around the kidney or in the other areas to which the pus has extended. The location of the abscess in the relation to the kidney naturally varies. The operative notes show that the abscesses may be found localized behind the renal pelvis, adjacent to the lower pole, adjacent to the upper pole, behind the kidney, or even anterior to it. Sometimes there are several small abscesses. Of course in the long-standing cases the breaking down and suppurating of the perirenal fat has gone on to such a point as to form a large abscess cavity that is easily broken into just under the muscles of the flank, and one has no idea what the original relation to the kidney was. In those cases where there is a large abscess and the kidney is sufficiently involved to make nephrectomy inevitable, it is usually advisable to be content to drain the abscess first and then do a secondary nephrectomy at a later sitting. In cases where the abscess is small, one can often do the inevitable nephrectomy at the same time the abscess is drained. The postoperative care is usually simple enough unless the patient is suffering from a spreading or generalized infection that may tax one's ingenuity to the utmost to save the patient.

In this series there were ten deaths, a mortality of 11.4%. Richardson's series had a mortality of 10.2%. Several of the deaths occurred in cases of long-standing urinary infection, or cases that were operated on by mistake for some supposed intra-abdominal pathology. Pyonephrosis, subsequent peritonitis or septicemia, or both, accounted for six deaths. Septicemia without known major kidney pathology caused three deaths, and bronchopneumonia accounted for the tenth death. Those cases that were autopsied all showed a definite renal focus for the perinephric abscess.

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PROLONGED BED REST TREATMENT IN PULMONARY TUBERCULOSIS*

BY PAUL DUFAULT, M.D.†

DETTWEILER was the first to keep the febrile consumptive on absolute rest and to prescribe rest on reclining chairs in the open air. Kretzschmar of Brooklyn (1888), Trudeau (1895), and Minor, inspired by his work introduced his method in this country, but the foremost advocate of intensive and prolonged bed rest is Dr. J. H. Pratt of Boston. As early as 1907 he started his Tuberculosis Class on the basic principle of prolonged bed rest. He carried on his work methodically, exercising much care and patience and never letting difficulties and rebukes discourage him. After ten years, he could compare his results advantageously with those obtained by work and exercise. He published numerous papers and articles. His doctrine caused much discussion and comment, but it was eventually accepted.

Acceptation, however, does not necessarily mean application when it comes to principles. Rest treatment was not yet adopted by the State Sanatoria, and Dr. Pratt could write in 1916: "The figures show that the end results of home treatment in the Emmanuel Church Tuberculosis Class were better than those obtained at Rutland."

The residents of sanatoria and those generally interested in the treatment of tuberculosis know the almost stereotyped story of the patient "sent to the country," advised to "take it easy" or to "hang around the house." To them, rest simply means absence from the store or from the mill. It permits a wide choice of pastimes such as riding, walking, swimming, playing golf, or anything within the reach of a leisurely young man about town. It includes light tasks "just to work up an appetite," like digging the garden or filling in the driveway for the new garage. Occasionally, from mere lonesomeness, the patient may lie on the couch in the kitchen for an hour or so. This picture may be regarded as overdrawn. Such cases, however, have repeatedly come within the scope of the writer's observations. One may say that patients are not likely to receive advice of that kind in our enlightened year 1932. They do, unfortunately, too often. When they are not explicitly told to "cure" in the above described manner, they are left without sufficient instructions. The meaning of the word rest is not carefully explained

*Read at the meeting of the American Sanatorium Association at the Middlesex County Sanatorium.

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to them and definite rules are not given. The patient's usual and natural reluctance in accepting his physician's advice on this particular point should be the most potent reason for the physician to be specific and insistent. The patient should be convinced first.

Conviction, however, cannot be projected into the consciousness of the patient unless it be deeply felt by the doctor. And is it? Is the tremendous value of rest worthily appreciated by the majority of physicians? One is inclined to doubt it, if one judges by the small number of patients who have been kept at rest, while waiting for their admission to the sanatorium.

If such is the attitude of the profession at large, what is the practice of those engaged in the field of tuberculosis? It is more in favor of bed treatment as a rule, but there are exceptions.

As late as 1927, John J. Russell of New York City discards sanatorium treatment altogether as too onerous and impractical and claims that "average patients with an evening temperature of 100 may continue their customary occupation without harm while being treated". The same year, Filip Sylvan, recommending gymnastic exercises, does not think that "a chronic high temperature, say 38 to 39 C., need be a contra-indication."

In numerous sanatoria, the period of rest treatment is still very short. Is it lack of conviction? Is it need of proofs?

In spite of the statistical evidence published in favor of prolonged bed rest, many physicians who are today treating pulmonary tuberculosis are still unconvinced that it is either necessary or desirable. A comparison of the Rutland State Sanatorium statistics before and after the application of rest treatment will bring out striking facts.

In 1916, Dr. Pratt, making a survey of the results of his rest method as applied in his Emmanuel Church Tuberculosis Class, could show the comparatively little value of sanatorium treatment, understood as it was in Rutland at the time (tables 1a and 1b).

In 1927, he demonstrated the after-results of prolonged bed rest (Emmanuel Church Tuberculosis Class); of little or no rest (Rutland 1911 figures); of combined rest and exercise (Midhurst, England); of work and exercise (Frimley); in the following graph:

Prolonged bed rest was instituted in the Rutland State Sanatorium in 1917. Dr. E. B. Emerson has long been a firm believer in rest treatment. Nine years ago, he voiced his opinion in the *Boston Medical and Surgical Journal* (September 13, 1923): "Without rest, all other known measures, important as they may be, will surely fail. It is the bed-rock of tuberculosis therapy . . ."

The most difficult problem confronting him was to carry his conviction to the patients, and to convert them to his belief. He knew very well that rules and regulations would fail lamentably

Table 1a

Emmanuel Church Tuberculosis Class Condition 1915

Admitted In	Number of Patients	Well and Working	Living	Dead	Not Traced	Percentage Well	Number of Years
1905	17	4	1	12		23.5	10
1906	27	9	1	17		33.3	9
1907	24	15	1	7		66.7	8
1908	22	14	2	4	2	63.6	7
1909	12	5		7		41.6	6
1910	19	10	1	8		52.6	5
1911	24	16	2	6		66.7	4
1912	24	15	1	8		62.5	3
1913	10	9	1			90.0	2
Total	179	98	10	69	2	55.3	

Table 1b

Rutland Sanatorium Condition 1911

Admitted In	Well and Able to Work	Living	Dead	Not Traced	Number of Years
1899	21.1%	.1%	54.0%	23.7%	12
1900	24.5%	.1%	46.8%	27.5%	11
1901	25.2%	.8%	48.4%	28.8%	10
1902	24.3%	1.4%	38.4%	35.9%	9
1903	20.5%	1.4%	37.2%	40.5%	8

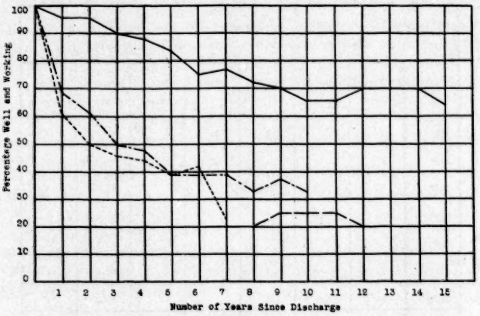
unless received in minds already prepared by understanding and good will.

Of late years physicians have consented to come out of their "sanctum sanctorum" and to address laymen in their own language. Educational health campaigns have been conducted through the media of various publications and periodicals, and conveyed by radio talks. Every newspaper has its daily medical column. As a consequence of this, the public has become health minded, and its attitude has changed. Patients are no longer content to accept dogmas and to obey orders. They want to know. They want to understand at least the principles of their treatment before submitting to it. Their comprehension and acceptance are the conditions "sina qua non" of their coöperation. And where is coöperation more necessary than in the treatment of pulmonary tuberculosis? This idea is well summarized in the paper of Dr. Emerson just quoted. "The first step in the treatment of the patient is his education, carrying home to him the truths he must know and must apply with all the zeal that he lives his religion. He must have something more than a generalized knowledge of the disease and its treatment. His ideas must be systematized and crystallized in so far as they apply to him personally."

Proceeding from that principle, instructions were begun individually and collectively. The patient heard his first talk on rest on the day of his admission, in a heart-to-heart conversation with the physician to whose service he was assigned. More was said to him on the same subject when he was examined before the staff a few days later. But the most important feature of all was the institution of a course of about thirty lectures given weekly during the winter months. The principles of rest treatment were patiently demonstrated and its mechanism carefully explained. Every question was answered and every objection was discussed.

Results did not appear immediately. It took some time for the idea to penetrate and take root. After a year or two, however, physicians and nurses began to notice that the hours of treatment were more faithfully kept, and even voluntarily lengthened.

The same policy of conviction has been carried out up to the present time with increasing success. The results are now, and have been already for some years, very tangible. Our patients may be seen on their beds at almost any hour of the day. Particularly remarkable is their observance of "Rest Period" (1:30-3:30 P.M.) which they are told to keep religiously, even after discharge, in the measure possible. There is not a stir to be seen or a whisper to be heard in the wards. The patients require perfect quiet of each other. Most of them are asleep, and those who are not know better than to disturb their wardmates. No supervision whatsoever is needed during that time.



GRAPH 1.

Emmanuel Church Tuberculosis Class ——
Frimley - - - - - - Midhurst - — - — - —
Rutland 1911 — —

Let us now come to more positive facts and examine the results obtained in the light of a short comparative statistical study. A survey of the 1922 group will furnish the data.

Excluding the non-tuberculous and the not considered cases, there were 359 patients discharged in 1922, 278 of whom could be traced. This return, representing 77 per cent of the whole group, is excellent if one stops to consider that the great majority of these patients were coming from and returning to large centers, especially Greater Boston. Knowing the frequency with which city dwellers change their place of abode, a ten year old address is rather a poor lead. Assuming that these returns were a fairly representative cross section of the whole group of patients discharged in 1922, we thus

procured the comparative percentage shown in table 2.

Well and working 8 to 10 years after admission — Emmanuel Church Tuberculosis Class (1917)	42.40%
Able to work 8 to 10 years after admission — Rutland Sanatorium (1911 Survey)	21.1% to 25.2%
Able to work 8 to 10 years after discharge—Rutland Sanatorium (1932 Survey)	41.7% to 37.7%

TABLE 2.
Percentage Comparison of Well and Working After 8 to 10 Years.
All Stages and All Conditions on Discharge.

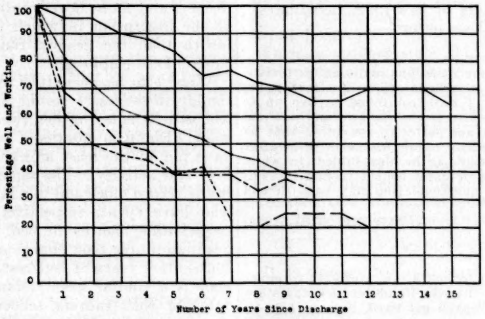
Let us now turn these figures into Graph 1, and consider the 1922 Rutland curve (Graph 2).

The 1922 Rutland curve still goes down more rapidly and remains below that of the Emmanuel Church Tuberculosis Class. The most logical explanation seems to lie in the difference in the patients themselves. The Emmanuel Class patients and those of Frimley and Midhurst were all able to work on discharge. The Rutland cases include not only the patients able to work, but all except those whose disease had not become quiescent. In addition, because of contracts with counties, all cases regardless of condition were accepted at Rutland. As a result, the majority of the patients were moderately and even far advanced. We must also remember that the Emmanuel class patients were constantly supervised, while the Rutland patients were not. As the years passed, they were bound to forget the advice received and the good resolutions taken. Besides, rest treatment was not yet what it could have been and what it became since, as illustrated in table 3.

A striking proof of the value of continued rest as a safeguard against relapse is furnished us by a survey of the Sanatorium payroll. Rutland has carried out from the beginning the generous and very humanitarian policy of employing ex-patients in the measure possible. Thus the oldest state sanatorium in North America has simply but effectively and lastingly done its humble share toward the practical solution of the vast problem of rehabilitation and sheltered employment for the tuberculous.

Time in Bed and Time in Sanatorium in 1922				Time in Bed in 1932	
Stage	Number of Patients	Average Time in Bed	Average Time in San.	Number of Patients	Average Time in Bed
I	48	51.9	299	9	156
II	98	70.2	326	49	259
III	132	115.9	346	38	427
Total	278	89.0	331	96	317

TABLE 3.
Number of days in bed and in Sanatorium in 1922 compared with the number of days in bed in 1932.



GRAPH 2.
Emmanuel Church Tuberculosis Class ——
Frimley - - - - - - Midhurst - - - - - - -
Rutland 1911 — — Rutland 1922 .....
These percentages are based as follows: Tuberculosis Class—bacillary patients with wage earning power restored; Frimley—bacillary patients who did five hours' hard labor before discharge without apparent ill effects; Midhurst—bacillary patients able to work on discharge; Rutland 1911—all Rutland admissions for the first five years of the Sanatorium's existence; Rutland 1922—bacillary patients quiescent on discharge.

Table 4 shows the number of years those pioneers have worked. It will be noticed that most of them had positive sputa, so that no doubt can be raised in regard to the diagnosis. The point I wish to bring out and to insist upon, however, is that very few had breakdowns.

What is the logical explanation? Certainly not light work and short hours. Our employees work as long and as hard as the average office or

mill worker, but they are spared the hardships of commuting; and as their living quarters are on the premises, they can and do rest after lunch. As the Sanatorium is at a fair distance from the nearest city, they are not tempted to share in so many social activities, and they spend most of their evenings quietly, reading or listening to the radio. The worry and extra work, which are so often synonymous with housekeeping among the less fortunate class, are also omitted from their lot.

That rest has been a life-saver, and that it still stands between them and any possible breakdowns, is the feeling of two 1922 "graduates", as expressed in these letters:—

"*Dear Doctor:*

In response to your questions, will say that since my discharge from the Sanatorium and taking my residence at H....., I have continued to live up to the same rules as exist at the Sanatorium, and am doing very well. I am perfectly convinced that as long as one *continues to follow the same rules* after leaving the Sanatorium as he was subject to while a patient, it will be of great benefit to him. I will gladly answer any questions you may care to ask at any time.

Yours very truly,

———."

"*Dear Doctor:*

I would like to write just a few words. I've never been so well before. I haven't had one sick day since I left there. I don't even get tired, because *I know now how to take care of myself. I can always find time to take rest hour, because I know what it means to me.* For several years after my discharge I was faithful in keeping up my treatment. I have had a physical examination every year, as you will see by my record. For the last fifteen months I have been under an awful strain. My husband, a war veteran, was taken sick in 1929. He was at the Veterans' Hospital for a year. No improvement was shown so he came home, and I nursed him here for fifteen months. He died August 10. I still cared for my home, also my outside work, but I always managed to take my quiet hour. I've always held my weight. I am always glad to answer any questions.

Yours very truly,

———."

The length of time during which we keep our patients in bed has more than once been found somewhat excessive. A considerable amount of ink has already flowed on the so-called drawbacks of prolonged rest, and numerous objections have been raised to its use.

One of the disadvantages often brought forward and greatly emphasized is that prolonged rest has a pernicious influence on the patients' morale; that it makes them lose their energy and even the ambition of being self-supporting. I do not know of any statistics on the subject, but I doubt that anyone engaged in the field of tuberculosis, or doing sanatorium work, has ever made such an assertion. A veteran in the army of tuberculosis workers, the Superintendent of the Rutland State Sanatorium, has had an aver-

Number of Years Worked	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	Sum
Number of Patients(Fem.	3	5	3	2	4	3	1	2	3	3	1	3		2	1	2	1			3	1			1	3		1		1	1		1	51
Number of Patients(Mas.	3	4				1				1	2	1	1					1		2	2			1	1	1				1			22
Total Number of Patients	6	9	3	2	4	4	1	2	4	5	2	4		2	1	2	2		2	5	1		1	2	4		1		1	2		1	73
Number of Breakdowns						1					1	1																					3

TABLE 4.
Statistics of Rutland Employees.

age of 50% bed rest "graduates" on his payroll for the past ten years. He has never found them neglectful, lazy, or undependable; on the contrary, he has noticed that they show a greater, deeper, and more hearty interest in the welfare of the institution in which they regained their health than the general rank and file of non-tuberculous employees.

Were I to be permitted to report a few particular instances, I would mention a secretary who is now entering her twelfth year of steady work. Eighteen months of intensive rest in bed have not in the least affected her mental alertness and activity. The same applies to a clerk in the record office and to a laboratory technician who have spent, respectively, twenty-one and twenty-nine months in bed. Six years ago, a graduate nurse took charge of a ward after five consecutive years of bed rest. The head laundry man is a similar case. Many of the patients, entering the training school after completing their treatment, have almost invariably proved to be excellent pupils and later very efficient nurses. Most of them occupy responsible positions in different hospitals and sanatoria throughout New England. Extremely valuable help was given me in the preparation of this paper by a patient still under treatment. He carefully computed the tables shown above and took so much interest in his work that, in spite of repeated warnings, he more than once exceeded the time limit during which he was allowed to work.

Whenever a tuberculous individual claims to have lost his ambition in the sanatorium, a careful scrutiny of his previous history will almost infallibly reveal that the supply he brought in was indeed scanty.

(NOTE—The author wishes to express his indebtedness to Daniel A. Robinson for his valuable help in preparing this paper and in tabulating the figures. The coöperation of Dr. Pope, Director, Division of Tuberculosis, in finding the cases made this survey possible.)

ECTOPIC PREGNANCY*

BY JOHN F. CURRAN, M.D.,† AND RAYMOND H. GOODALE, M.D.†

NO more dramatic surgical problem presents itself to the operator than that of ruptured ectopic pregnancy. On the other hand, perhaps no case which before surgery is so seriously ill makes such a brief and uncomplicated convalescence after timely operative treatment. Despite the fact that the diagnosis appears to be obvious, it is not infrequently missed, and death follows because of deferred operation. These facts have prompted us to present for your consideration our observations in the analysis of the records of 108 consecutive cases of this type which occurred during the past ten years at the Worcester City Hospital.

HISTORICAL

While this condition is not new, science has seemingly made little progress in the treatment of such cases. In early Biblical times we read of cases, as told in the Talmud, which were ectopic cases in the light of modern science. Medical history makes mention of them in the eleventh century. Quoting Rutherford[1]: "Ectopic pregnancy was unknown to the ancients. The first case reported is that of an Arabian physician in Spain about the middle of the eleventh century. He reports a case wherein he saw parts of a foetal body escaping from the abdomen of a woman by the process of suppuration. He found that the foetus had died in the uterus of the patient and an abscess later formed at the navel, and it was from this abscess that the foetal bones were extruded." There was also a famous case of a petrified embryo reported by Cordaeus[2] in the sixteenth century called "The Lithopaedian of Sens", which was of this same type, as were practically all of the earlier reported cases.

Riolamus[2], in 1604, was the first to describe definitely a fetus found in the Fallopian tube. The first case of surgical interference for the removal of the fetus was by Primerose[3] in 1594. This woman had two extra-uterine pregnancies. The first evidence of a true understanding of the cause and the phenomena of extra-uterine pregnancy was found in the works of Pièrre[4] published in 1718.

The first authentic case of operation for extra-uterine pregnancy in America was reported by Dr. John Bard[5] a surgeon of New York in 1759. This operation was performed upon this side of the Atlantic for the second time in January, 1791, by Dr. William Baynham[5] of Virginia, the subject being the wife of a Virginia planter. Dr. Baynham was a country physician, and the operation was successful. He performed a similar operation in 1799 which also was successful.

Lawson Tait[6], in 1883, more than two centuries later than the time of the operation by Primerose, was the first to operate deliberately on a case of ectopic rupture, as distinguished from unruptured ectopic gestation. About this time the use of electricity in such cases was advocated for the purpose of destroying the life in the ovum by means of a strong galvanic current applied to the tube affected, through the vagina. However, as a result of the reports by Tait of forty consecutive operations for tubal gestation, with but a single death, operation was generally recognized as the preferred treatment in such cases.

It is interesting to note that up to 1876 ectopic pregnancy was considered a pathological curiosity which had never been noted at many leading hospitals.

Various authors state that the incidence of tubal gestation is by no means rare, and the results depend very largely for their success upon early discovery of the condition. The estimations of the frequency of occurrence of ectopic pregnancy have differed. Schumann[7] made an analysis of all the pregnancies reported in Philadelphia in one year, and it was estimated that there was about one ectopic in every three hundred cases.

ETIOLOGY

It is undisputed that any factors which delay or arrest the passage of a fertilized ovum in its journey from the Graafian follicle to the uterus, or cause it to become imbedded in any place other than in the uterus, may cause an ectopic pregnancy. This statement could be amplified in considerable detail, but we shall here cite only a few of the direct causes of this false implantation:

(1) Malformations inside the Fallopian tubes, which cause obstructions to the passage of the ovum. (2) Causes on the outside of the Fallopian tubes, namely contracting or kinking of the tube, resulting in the obstruction of the passage of the ovum.

Quoting Brady[8] "Usually these are due to a former Neisserian infection, although occasionally they result from an old ruptured appendix. . . . Occasionally tumors of the uterus or ovaries press on the tube and occlude its lumen. In one of my cases I was able to show that the further course of the ovum was stopped by a parovarian cyst, which so pressed on the

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Fallopian tube as to kink it sharply and occlude the lumen.

"It is evident that there are many factors capable of bringing about an ectopic pregnancy. Hahn[9], from an investigation of 240 cases from the Vienna Hospital in 1903, reached the conclusion that gonorrhea is the most common cause and stated that in large towns, where this infection is more prevalent, more cases of tubal gestation occur."

Other types of salpingitis may cause the formation of pockets with blind ends, in which the fertilized ovum becomes lodged. Another significant element in the causation of this condition, according to Dr. A. Miles Taylor[10], is tuberculosis, which by thickening and hardening the tubal walls prevents the free passage of the ovum. In the many specimens examined in our laboratory the combination of tuberculous salpingitis with ectopic pregnancy has never been observed.

The last word in the etiology of ectopic pregnancy has not been written, as evidenced by recent experimental work of Manzi[11]. He experimented in vitro on the action of the follicular fluid and of extracts of corpus luteum on tubal motility. Under the influence of the follicular fluid, he observed an increase of kinetic activity of the tube, manifested by a greater height of the contractions and a more regular rhythm. Under the influence of the extract of corpus luteum, on the other hand, he noted a decline or the almost complete disappearance of this activity. He states also the belief that what he observed experimentally will likely be confirmed in vivo, viz., the motility of the tube would be reinforced during the follicular phase (maturity, and, especially, rupture of the follicles), and would be regulated to favor the migration of the ovum; whereas, during the luteinic phase, the motility of the tube would be notably reduced, and likewise the motility of the uterus, to favor the implantation of the ovum. In pregnancy, the motility of the tube is reduced, as opposed to the condition during labor. In both these periods, however, the tube responds to the action of the two ovarian secretions in the same manner as in the experiments.

Possible sites of ectopic pregnancy: Statham and Shepherd[12] state that the varieties of implantation in their series of 100 cases were as follows:

Primary ovarian pregnancy	1
Secondary abdominal pregnancy	3
Tubal implantation	95
Pregnancy in an undeveloped horn	1

"True tubal pregnancy is much the commonest type of ectopic gestation. The implantation can be interstitial, isthmic or ampullary. The average reported frequency is roughly, interstitial 3 per cent, isthmic, 20 per cent, and ampullary 77 per cent." The location of the pregnancy depends upon the point of implantation of the ovum.

Interstitial pregnancy: It is essential to give the greatest consideration to interstitial pregnancy to which is attributed the highest mortality, doubtless because of the greater difficulty of diagnosis. Some assert that the tube in the interstitial portion is so narrow that consequently this would be the most common form of tubal gestation. But, fortunately, it is the least common, only one to three per cent of these cases being of this type. It is a condition where the impregnated ovum becomes implanted in that part of the tube which penetrates the wall of the uterus and undergoes development there. From the time the ovum makes its attachment on the wall of the tube the latter becomes enlarged and hypertrophied on account of an overgrowth of the tissues which constitute it, together with the infiltration of fluid between the cells of which it is composed. The blood vessels are increased in number and size and become extremely tortuous. The hypertrophy of the tube which occurs at the proximal extremity serves to constrict the opening more and more as the condition progresses until finally the cavity is closed as is the canal of the cervix in normal pregnancy. As the impregnated ovum develops, the wall of the tube becomes stretched and thin, and thus the strength of the wall is materially decreased.

Broad Ligament Pregnancy: Occasionally tubal rupture may occur through the portion of the tube not covered by peritoneum and the fetus is pushed downward between the leaves of the broad ligament instead of into the peritoneal cavity. The pregnancy may then continue in the broad ligament, but more frequently the fetus dies and a broad ligament hematoma is formed. Diagnosis in these cases is difficult.

Abdominal Pregnancy: This has been known to occur, though some authors express doubt that such a case can develop. This type, however, is rarely encountered.

Ovarian Pregnancy: This condition is generally believed to result from the fertilization of an ovum before it has escaped from the Graafian follicle.

SYMPTOMATOLOGY

Quoting Meaker[15], "The classical picture of early ectopic pregnancy is so well defined that one might reasonably expect the diagnosis to present few difficulties. The history includes a missed period, followed by spotting and staining; the concomitant mammary, gastric, and vesical symptoms of pregnancy; and abdominopelvic pain. Examination reveals slight enlarge-

ment and softening of the uterus, and a tender unilateral adnexal mass. . . .

"The early diagnosis of extrauterine pregnancy is often beset with difficulties. There are two general sources of confusion: (1) any or all of the classical symptoms and signs of ectopic pregnancy may be presented by other conditions; and (2) some of the classical symptoms and signs are absent or masked in a considerable proportion of ectopic cases.

. . . . . .

"Practical Conclusions.—1. Early extrauterine pregnancy presents a variable clinical picture, often atypical, and sometimes closely simulated by other conditions.

"2. One should always suspect extrauterine pregnancy when a woman in the childbearing age exhibits a menstrual disturbance of short duration and pelvic pain of recent origin.

"3. In the suspected case the closest attention should be given to the character of the pain. Associated faintness is almost pathognomonic.

"4. If obvious acute inflammation is not present, then any great pelvic tenderness, aggravated by movement of the uterus, is highly suggestive of extrauterine pregnancy.

"5. In the early case a short period of observation often serves to clarify the diagnosis."

According to Cadwaller[13], "Ectopia covers but a few weeks. . . . It is ushered in by cramps and ends in rupture of the tube. The rupture may be gradual or sudden, calling for speedy operation, often demonstrating itself by Cullen's sign, which is a bluish discoloration below the umbilicus."

As long ago as 1890 shoulder pains were recognized by John Ferguson[14] as indications of ruptured ectopic pregnancy and frequently since then they have also been recognized by other investigators. These pains may be described as painful sensations beginning on the apex of the shoulder and extending up into the back of the neck. Thus it is apparent, though science today is not so familiar with this subject as it might be, that shoulder pain is not a newly discovered symptom indicating ectopic rupture.

One symptom is constant,—pain. However uncertain the other symptoms may be in extrauterine pregnancy, pain is always present. In the early cases, while the ovum is still within the tube, the pain is a conspicuous symptom and has fairly distinctive characteristics. It is irregular in occurrence, severe in degree, sharp in character, and often radiates to the shoulders, the back, or the thighs.

DIFFERENTIAL DIAGNOSIS

Intra-uterine pregnancy with suspected abortion is characterized by profuse vaginal bleeding which is bright red in color. In extra-uterine it is usually slighter, dribbling, and brownish-red. The pain of abortion is that of typical intermittent cramps located over the uterus, gradually increasing in intensity, but in ectopic pregnancy the cramps are severe at the onset, very sharp and stabbing in one side. If there is much blood in the cul-de-sac, there is also pain on defecation. The cervix is usually softer in normal pregnancy, and it is not painful on movement. There is no fever, leucocytosis or tender mass.

Salpingitis: A history of recent gonorrheal infection, positive smears, gradual onset of pain, purulent vaginal discharge, higher fever, greater leucocytosis, no enlargement or softening of the uterus, hardness of cervix, and a less circumscribed mass and less intensity of tenderness all favor a diagnosis of salpingitis.

Pelvic Tumors: Ovarian cysts are characterized by their larger size, lack of great tenderness and absence of uterine enlargement, absence of cervical softening and of pain on moving the cervix. Small ovarian cysts may be more easily confused, but here the history and pelvic examinations should serve to differentiate the two. A fibroid at the uterine horn rarely causes confusion because of the history of the case and the hardness of the mass.

Appendicitis: This diagnosis can usually be made by the history and the pelvic examination. The pelvic examination would not show softening of cervix and pain on movement. The uterus would not be enlarged, and there would not be a bulging cul-de-sac.

Treatment: The recognized treatment of this condition was followed in all of our cases, namely, excision of the affected tube with supportive treatment in the form of saline infusion or blood transfusion when either was deemed necessary.

It is proper at this point to mention the method of Löhnberg[16] in his treatment of ruptured ectopic pregnancy with severe hemorrhage. He is most successful with his transfusions, a method which is more effective in helping the patients exsanguinated from ectopic ruptures. Löhnberg's method is to extract the escaped blood of fresh tubal ruptures, and, when thoroughly filtered, to infuse it intravenously. He cites fourteen cases in which he was highly successful. This seems a useful method in lowering present mortality rates especially when time does not permit obtaining a transfusion donor.

Wallace[17], in a recent article, contends that while it has always been the advice of our best medical men to operate as soon as diagnosis of ectopic conditions is made, nevertheless the right to live should be given many of these little children who are merely started wrong. This he would accomplish by a system of transplantation, a feat which he maintains is not difficult in these days of advanced surgery. But little danger is entailed and the child develops and is born as was the intention before obstruction. This can be done if an early diagnosis is made.

The uterus advances with tubal gestation and, in fact, takes on all the early features of normal pregnancy. It would, therefore, seem possible to transplant the product of pregnancy into the uterus, where everything is ready and waiting for it.

Dr. Wallace reports a case where he operated upon a woman for a uterine cyst, and, in the course of the operation discovered a tubal pregnancy which he removed intact. He fastened it to the endometrium of the uterus, where it became attached and grew to maturity, and a normal child resulted. Thus it can be seen that transplantation upon early diagnosis has been successful, and should be recommended in appropriate cases.

ANALYSIS OF 108 CASES

The hospital records of ectopic pregnancies from 1922 through 1931 were reviewed and analyzed. It is hoped that the data thus obtained will give us some lessons whereby we may improve our work in the future. There were 7064 maternity cases in this hospital during the years 1922 through 1931. During this time there were 108 ectopic pregnancies, making the incidence three ectopic per 200 completed uterine pregnancies.

Symptomatology: Pain was present in all of our cases. It is the constant symptom in ectopic pregnancy. The location of the abdominal pain was as follows:

	Number of cases
Left lower quadrant	14
Right lower quadrant	28
Lower abdomen	25
Generalized abdominal pain	29
Epigastrium	2
Right upper quadrant	1

The special locations of pain were the following:

Rectal pain on defecation	6
Pain on urination	4
Pain in back	1
Pain radiating to shoulders	2

Menstrual alteration was found in all cases. This included increased bleeding, decreased bleeding and absence of menses from three to twelve weeks. Sixty-nine patients complained of irregular vaginal bleeding; in the remaining thirty-nine there was history of cessation of menses. The duration of pregnancy averaged 5.7 weeks.

Additional symptoms were seen as follows:

	Number of cases
Fainting	12
Nausea	10
Vomiting	21
Weakness	4
Chills	2
Difficulty in breathing	1
Dizziness	1

Signs: Analysis of abdominal findings:

	Number of cases
Mass in one of lower quadrants	5
Tenderness in one or both lower quadrants	20
Generalized abdominal tenderness	5
Abdominal spasm	9
Fluid in abdomen clinically	7

Analysis of pelvic findings:

Cervix softened	12
Pain on moving cervix	108
Fullness and tenderness in one of vaults	26
Tenderness only, in one or both vaults	15
Mass only, in one vault	14
Uterus enlarged	3

Because Polak believes that repeated pregnancies per se may be a contributing factor in the etiology of ectopic pregnancy, we present an analysis of the pregnancy status of our cases. There were 22 patients who had no previous pregnancy and 13 in whom pregnancy could not be determined from the records. The following tables show the various previous pregnancies in our series:

Patients	Normal Previous Pregnancies
2	6
7	5
7	4
6	3
18	2
16	1

Patients	Previous Abortions
1	1

Patients	Previous Stillbirths
3	1

Patients	Previous Premature Births
1	1

Patients	Previous Ectopic Pregnancies
4	1

The oldest patient was 44, the youngest was 19, and the average was 30.7 years.

Ninety-nine patients were married, seven were single, one was divorced and one a widow.

Only one colored woman was found in this series, but the incidence is said to be the same in both races, in hospitals where equal numbers of white and colored people are treated.

There was a total of 11 previous lower abdominal operations which included appendectomies, salpingectomies, and oöphorectomies. There were four patients who had had a previous dilatation and curettage.

The following table shows the preoperative diagnoses:

	Cases		Cases
Ectopic pregnancy	89	Ovarian cyst	1
Surgical abdomen	3	Fibroid	4
Appendicitis	1	Endometritis	1
Metrorrhagia	1	Salpingitis	2
Pelvic abscess	1	Threatened miscarriage	3
Retroverted uterus	1		

The tubes were found ruptured in 94 and unruptured in 13 cases. One ovarian pregnancy was unruptured. There were 57 pregnancies in the right tube and 50 in the left tube.

The locations of pregnancies were as follows:

Right tube	Cases
Ampulla and fimbriated end	53
Ampulla	1
Isthmus	3
Left tube	Cases
Ampulla and fimbriated end	39
Ampulla	4
Isthmus	1
Ovarian pregnancy	1

Following are the complications:

	Cases
Infected wound	4
Intestinal obstruction with ileus	2
Pelvic abscess	2
Postoperative hernia	2
Phlebitis with pulmonary embolus	1
Sepsis of transfusion wound	1
Mucous colitis	1

Mortality: There were six deaths in the 108 cases. Following is a discussion of these deaths:

CASE No. 1. (Non-operative case.) A housewife, 30 years of age, admitted to the hospital in profound shock, and died before surgery could be attempted.

CASE No. 2. (Operative case.) A housewife, 34 years of age, came in with a ruptured ectopic pregnancy. Operation was delayed by refusal of husband to accede to our advice. Transfusion was given at operation. Patient died on fourth day, of postoperative ileus.

CASE No. 3. (Operative case.) A housewife, 33 years of age. A pre-operative diagnosis of ruptured ectopic pregnancy was made and a salpingectomy was done. The day after operation the patient became restless and vomited greenish material once. Patient continued to be restless, vomited several times, abdomen became distended and patient went into collapse, dying on the seventh postoperative day, of paralytic ileus.

CASE No. 4. (Operative case.) A housewife, 35 years of age, admitted to the hospital on the genitourinary service because of vesical symptoms. She had a two day residence before being transferred to the surgical service, where she was diagnosed as ruptured ectopic, quickly operated upon and transfused, but failed to rally.

CASE No. 5. (Operative case.) A housewife, 37 years of age, who was pulseless on admission and had a blood pressure of 58/36. She was diagnosed as ruptured ectopic pregnancy, and an operation was done as a last resort. During the operation intravenous normal saline solution was given, and donors were typed for transfusion. Immediately upon return to the ward, intravenous of normal saline was started and stimulants were used, but the patient succumbed.

CASE No. 6. (Operative case.) A housewife, 42 years of age, died of a pulmonary embolus on the eighth post-operative day. At autopsy a thrombus was found in the left common iliac vein with a terminal infarction from an embolus in the right lung.

Of these deaths, two died of paralytic ileus, one of thrombosis of the left common iliac vein with lung infarction, and two as the result of delayed surgery. Of the two latter deaths one occurred where evidently a severe hemorrhage had taken place before entrance to the hospital, and surgery was attempted as a last resort. In the other case the patient had a hospital residence of two days before diagnosis was made and again surgery was attempted as a last resort.

These five operative deaths make a mortality rate of 4.6% of the ectopic pregnancies. We feel that surgery ought to be held responsible in but three of these deaths, namely, the two cases of paralytic ileus, and the infarction of the lung. In the other two, surgery was done as a last resort without much hope of success. The corrected mortality would then be 2.8 per cent.

COMMENT

This discussion would not be complete without mentioning the laboratory aids in diagnosis. The advent of the Aschheim-Zondek test gives us a real aid in the diagnosis of pregnancy but this procedure requires at least twelve hours, and many times the diagnosis of ectopic pregnancy cannot be delayed. Wilson and Corner[18] cite six cases of operated ectopic pregnancy of which three gave a positive test and three a negative test. The test remains positive only as long as living placental tissue is in biological contact with the maternal blood.

The use of the van den Bergh test as described by Dumphy and Fallon[19] is a laboratory aid which has more practical value. A positive delayed direct reaction indicates the presence of free blood. This is a simple test and can be performed in a few minutes.

It seems advisable here to explain the symptomatology on the basis of the underlying pathology. This cannot be done better than by quoting Polak[20].

"The uterus is enlarged, because it contains a decidua which was previously prepared for the reception of the ovum. The cervix is soft, due to the congestion consequent upon pregnancy. This symptom is variable and is unimportant from a diagnostic standpoint. On bimanual examination the uterus does not have the characteristic diagnostic sign of pregnancy, —that is to say, the elasticity of the median portion of its anterior wall and the compressibility of its isthmus. The absence of these changes in consistency is due to the absence of the growing ovum in the uterus which, although enlarged, is not changed in shape or consistency save for the slight softening of the cervix which is not constantly present. The cervix is very sensitive to motion. This is shown by palpation and is due to the peritoneal irritation from the blood which gravitates from the end of the tube or through the tubal wall because of its porosity

and finally to the prolapse of the tubal mass into the cul-de-sac.

"This reaction of the peritoneum covering the uterosacral ligaments makes them sensitive; hence whatever moves these sensitive bands will cause great pain. This sign (pain on movement of the cervix) was present in all of Polak's cases. The pulsation of the uterine artery is more apparent on the side where the gestation sac is found. This sign is particularly noticeable after intraligamentous rupture, because the blood supply is increased on the side of the pregnancy and the caliber of the vessel is enlarged; hence because of the tendency of the pregnant tube to drop into either the posterior or posterior-lateral cul-de-sac, the artery is depressed and is brought more within the reach of the finger than is the case in normal pregnancy. The uterus is displaced because the tubal tumor, owing to its weight, has fallen into the lateral, or the posterior or the anterior cul-de-sac. Therefore, the greater the bulk of the tumor, the greater the displacement of the uterus; obviously the uterine displacement is less apparent in early unruptured ectopics with little blood accumulation in the cul-de-sac.

"The tumor displacing the uterus has certain definite characteristics; it is of rapid growth. This increase is due not only to the growth of the ovum but to the extravasation of blood which takes place into the muscle and decidua in the tube and thus increases the size of the gross mass. It is sensitive because the tubal covering is stretched to its utmost. It is fluctuant because the contents of the ovum and the contained blood are fluid. When ruptured or tubal abortion occurs, there is sudden pain, due to the erosion through the tubal wall and escape of the ovum, or to the expulsion of the ovum from the ampulla. The expulsion is due not only to peristalsis but to the formation of blood clots within the tube. The peritoneum immediately reacts and there are signs of intra-abdominal calamity, abdominal pain and shock."

SUMMARY

This article consists of an analysis of 108 cases of ectopic pregnancy taken from the records of the Worcester City Hospital. We wish to emphasize the importance of a careful history and physical examination. A review of our cases shows that all gave a history of menstrual irregularity with abdominal pain, ranging from colicky to knife-like in character. All had pain on moving the cervix. This symptom complex was consistent in all of our unruptured cases. The ruptured cases presented *in addition* the signs of internal hemorrhage such as weak pulse, pallor, sweating, bulging in a cul-de-sac, and occasional shifting dullness in the flanks.

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THE HEREDITARY ASPECT OF ARTERIOLAR (ESSENTIAL) HYPERTENSION*

Study of Three Generations of a Family

BY DAVID AYMAN, M.D.†

THE etiologic relationship between heredity and arteriolar (essential) hypertension is shown by clinical experience[1], by the study of family histories[2], and by the reports of families in which there is a high incidence of the disease[3].

The study of the family presented in this paper differs from the above studies in several respects. Every member of three generations of this family is alive. Therefore, instead of relying on family history for evidence of cardiovascular disease in some members of the family, every person in the family above the age of six has had his blood pressure determined by the author personally. In previous reports only

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one blood pressure reading is given for each person. In the present study, every person who had abnormally high readings had his blood pressure checked on another day, and in many cases on four or five separate days. Finally, the present family appears to be the first reported in which a tendency to arteriolar hypertension is shown by readings of the blood pressure to be present in three successive generations.

REPORT OF FAMILY

Referring to figure 1, it is seen that this Russian-Jewish family consists of thirty-two members, excluding the mates of the second generation. The members of the first and second generations were born in Russia and came to Boston 20 years ago. The mates of the second generation have been studied as controls. The blood pressure readings, age, sex, height, and weight of each person in the family are presented in Table 1. Using Rogers and Hunter's[4] standards of average blood pressure, it is seen from figure 1 and table 1 that 100 per cent of the first generation, 80 per cent of the second generation, and 25 per cent of the third generation had abnormally elevated blood pressure readings on both first and second visits. One member of the second generation (patient I) had elevated readings only on the first visit and was not included in these figures. Patients B, F, and J had known previous to my findings that they had elevated blood pressures. In striking contrast to the above findings, 100 per cent of the mates of the second generation had normal blood pressure readings.

No attempt was made to study the body build of the members of the family. As seen in table 1, the two definitely obese members (patients D and M) had arteriolar hypertension, but there were just as many obese persons among the mates with normal blood pressure readings. It is to be noted, however, that patient H, one of the two non-hypertensive members of the second generation, was the thinnest member of the family, being twenty-eight pounds below the average weight for his height and age.

The author had no opportunity to make physical examinations except in patient A of the first generation. This person showed left-sided cardiac enlargement (confirmed by roentgenray), and moderate narrowing of the arterioles in the fundus oculi. Further studies of this patient disclosed a gastric carcinoma. On subsequent examinations of her blood pressure during the development of cachexia secondary to her advancing carcinoma, her blood pressure became lower (table 1).

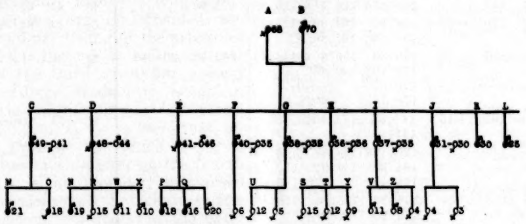

FIG. 1. Arteriolar hypertension in three generations. • indicates the members having hypertension; O indicates those with normal blood pressure readings. The numbers beside each of these symbols indicate age. The letters above each symbol identify each person, referring especially to table 1, in which blood pressure readings of each individual in this family are given. The parents of patient B died at 85 years of age, the cause of their deaths being unknown. The history of the parents of patient A is not available.

A few observations were made concerning the personality characteristics of the family. In general, most of the mates agreed that they had married into a high-strung family. They also agreed, however, that patient H, a non-hypertensive member, was decidedly different from his brothers and sisters, in that he was more of the happy-go-lucky type. Patient B of the first generation, always intensely religious, has spent the greatest part of his days in the synagogue. His wife, patient A, was, therefore, obliged not only to rear a family of ten children but to provide for them. This she successfully accomplished. Most of the members of the second generation were physically very active. Although without education, they made great and successful efforts in the dress manufacturing business. Despite recent heavy losses, their energy and efforts to retrieve continue unabated. At their work, they are considered extremely fast. Emotionally the majority of them are high-strung, being quick-tempered and sensitive. Many of these characteristics are already evident in the hypertensive members of the third generation. In general, the majority are of the personality type I have found unusually frequent among hypertensive patients[5].

TABLE 1

BLOOD PRESSURES, AGE, SEX, HEIGHT, AND WEIGHT OF 26 MEMBERS OF A COMPLETE FAMILY OF THREE GENERATIONS (AS DIFFERENTIATED BY THE HEAVY LINES) AND OF 8 MATES OF THE SECOND GENERATION

Patient	Sex	Age	Height in inches	Weight in pounds	Pounds above or below average normal	Blood pressure readings, systolic and diastolic: First visit	Second visit	Third visit	Fourth visit	Fifth visit	*Mates*: Sex	Age	Height in inches	Weight in pounds	Pounds above or below average normal	Blood pressure readings
A	F	68				198:90	152:74	146:58 130:58	160:70	140:60						
B	M	70				184:82 164:86	194:90 194:90	188:88	164:66 168:70							
C	M	49	61	141		218:134	220:118	170:90	178:90		F	41	63	180	44	104:64
D	F	48	62	170	+34	166:92 166:86	164:80 162:80	118:78			M	44	64	132		120:70
E	F	41	60	125	— 3	180:80 178:80 170:80 170:76	150:80 146:80	146:58	200:90	208:82 182:94	M	46	61	128	8	114:74 110:76
F	M	40	66	165	+15	180:98 180:98	174:100				F	35	"Obese"			106:68
G	M	38		140		180:100 164:98 176:100	148:88 142:92	140:90 152:92 140:86			F	32	64	135	3	128:78 130:78 126:78
H	M	36	64	112	—28	136:86 130:90 122:88	138:94	110:80	126:88		F	36	63	170	38	110:70
I	M	37	59	135	+ 9	152:88 148:88 146:90	136:86 136:86				F	35	63	151	19	124:78
J	M	31	62	126	— 4	180:90 160:98 170:90	148:88 140:88	158:88			F	30	61	115	8	134:74 130:78
K	M	30	64	130	— 6	150:90 152:92 150:94	134:84 144:90 146:92									
L	M	25	63	123	— 6	144:82 140:86	130:78 132:80 124:76	150:90 146:86	128:80							
M	M	21	66	207	+68	144:80 140:84	160:90 158:90									
N	M	19	68	160		144:76 134:72 124:68	150:70 132:70 124:60	140:80 136:88	124:70 110:70							
O	F	18	61	118	+ 2	140:80 130:84	138:86 136:82									
P	M	18	65	138	+10	140:80 150:80 142:72	142:70 140:68 140:68									
Q	M	16	67	122	—14	130:68 128:68 132:78	150:84 146:72 156:68									
R	F	15		140		120:72 116:80	118:70									
S	F	15		110		120:72	134:70 128:70	120:76								
T	F	12	58	82	+15	118:60	118:78									
U	F	12	57	92	+10	140:60 134:50	112:60	128:58								
V	M	11				124:86 114:86	112:70	128:72 116:72								
W	M	11	53	84	+17	124:70 126:64										
X	M	10	50	64		126:60 118:60 100:70										
Y	F	9	52	60	— 4	112:58 106:58	114:68									
Z	M	8				114:52	108:50									

COMMENT

It seems clear that in this family there is a marked tendency to arteriolar essential hypertension. That excitement associated with the determination of blood pressure is not the fundamental cause of the hypertension is indicated by the findings in the eight mates; for, although their blood pressure determinations were made under exactly the same conditions as for the family, in each instance they had normal blood pressures on the first reading. It is to be noted also that in cases B, F, and J, abnormally high readings had been found on previous occasions by other physicians. In patient A, the blood pressure drop on later visits was associated with the cachexia of malignancy; and, further, this person had evidence of long-standing hypertension in the form of left-sided cardiac enlargement and narrowing of the arterioles in the fundus oculi. Patient D had a normal blood pressure on the third visit, but normal blood pressure readings frequently occur in mild or early arteriolar hypertension[6]. Furthermore, referring to case E, it is seen that although the blood pressure was much lower on the third visit, it was much higher on the fourth and fifth visits. It is this tendency to marked variations from visit to visit that characterizes arteriolar hypertension, especially in the early stages[7]. It is to be noted in the second and third generations that the level of hypertension in each member increases with age. This is evidence that the hypertension begins in early life, that it rises with advancing age, and therefore the mild hypertension in the third generation probably indicates early arteriolar hypertension. If we compare the blood pressure readings of the normal mates with those of their hypertensive children, it is seen that the children already have not only higher systolic but also slightly higher diastolic readings than their normal parents. Interpretation of the blood pressure readings of those under thirteen years of age has not been attempted, as the average normal blood pressure of very young persons is not sufficiently well known.

SUMMARY

A tendency to arteriolar hypertension is demonstrated in three successive generations. In the third generation the hypertension has already appeared in the second decade of life. In this family, at least, arteriolar hypertension appears to have a strong hereditary tendency.

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DIVERTICULOSIS AND DIVERTICULITIS OF THE COLON*

BY S. ALLEN WILKINSON, M.D.†

In the past thirty years attention has been called with increasing frequency to diverticula of various parts of the alimentary tract. Practically any part of the tract is subject to these outpouchings which Moynihan[3] describes as "a wayside shelter by a main path". By far the greater number occur, however, in the colon, and of colonic diverticula, more than three-quarters are found in the descending colon and sigmoid.

ETIOLOGY

The first pathologic description of diverticula was by Cruveilhier[1] in 1849. In the next fifty years they were mentioned occasionally by anatomists, but no particular emphasis was placed on them. In 1899 Graser[2] propounded a theory as to their etiology which still has many advocates. He noted that most of the diverticula found at autopsy originated near a blood vessel and felt that the entrance or exit of these vessels through the muscular coat of the bowel created a potential weak spot. When conditions were favorable, a hernial protrusion occurred at these weak areas, burrowing along the blood vessel sheaths. Other investigators, failing to agree entirely with this theory, point out that diverticula occur at any point on the bowel, that more occur along the antimesenteric border than along the mesenteric attachment. Telling[4] felt that some abnormal congenital arrangement of the muscularis was responsible, while Beer[5] believed that muscular deficiency, resulting from constipation and age, caused the condition. Me-

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Grath[6] lists a portion of the following conditions which were held by various writers to cause diverticula: (1) obesity, (2) cachexia, (3) previous obesity with subsequent loss of intra-abdominal fat, (4) normal structure of the intestine, (5) intraintestinal pressure, (6) relation of diverticula to points of entry of vessels through intestinal wall, (7) location of epiploic appendages, (8) infection involving lymph channels along blood vessel walls, (9) muscular deficiency, (10) congenital weakness of muscularis, (11) constipation, (12) old age.

PATHOLOGY

There have been several anatomic or pathologic classifications of diverticula, the commonest one dividing them roughly into true or false diverticula, depending on whether they contain all the coats of the bowel or whether one or more coats are absent. Clinically, any such classification is of little value, as there is no distinction to be made in the symptoms produced by the various types.

Diverticulitis has been well described by numerous writers, and it is not the purpose of this paper to add anything new to the subject. Wilson[7] has described a condition which he terms peridiverticulitis. In this condition the inflammatory reaction surrounds the diverticulum but does not involve the mucosa. McGrath[6] emphasizes it thus: "The most constant pathologic process, and an essential in dealing with infection through these diverticula is the chronic proliferative extra-mucosal inflammation." He also emphasizes the fact that cancer is apt to be associated with diverticulitis in a large proportion of cases (25% in his series). W. J. Mayo[8], Wilson[7], Jones[9], and others have all called attention to the frequency of cancer associated with diverticulitis. Not all writers concur in this, however. Eggers[10] and Erdmann[11] are of the opinion that cancer is a coincidental disease. They quote statistics to show that cancer of the colon is as frequent in cases without diverticula as in those where they are present.

SYMPTOMATOLOGY

Practically all writers are in agreement that until one or more diverticula become inflamed, the condition is symptomless. Erdmann[11] says of diverticulosis that it "is a well established disease, at least roentgenologically if not symptomatically, and requires but the onset of a pathologic process of one or more of the protrusions to become a definite clinical entity."

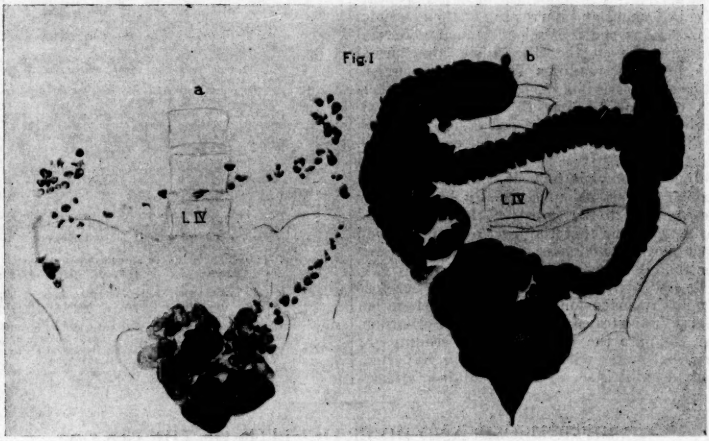

FIG. 1. Diverticulosis:
(a) Twenty-four hour film showing diverticula throughout colon.
(b) Opaque enema (same case) showing a few scattered diverticula visible, remainder obscured.

If inflammation has become established, the symptoms are often not so obvious as many writers lead one to suspect. In the milder cases there is usually pain near the umbilicus or on the left side. Frequently there is a low grade fever and a moderately elevated leucocyte count. Constipation is the rule, although intermittent diarrhea and constipation are common. Nausea, gas and shifting abdominal cramps are almost always present.

In more severe cases, in addition to the symptoms mentioned above, there may be sudden attacks of obstinate constipation, sometimes

amounting to complete obstruction, but usually followed by a watery diarrhea. Frequently there is a palpable tumor in the left abdomen. Because of the tendency to abscess formation, perforation into another viscus, particularly into the bladder, is not rare. Hemorrhage from the bowel is uncommon but does occur.

DIAGNOSIS

Since diverticulosis is symptomless, the diagnosis can be made only by the roentgen ray. No one roentgenologic technic suffices to show diverticula when they are present. The combination of a film taken twenty-four hours after a mouth meal together with a film of an opaque enema showing the bowel distended and a film showing contraction after evacuation of the enema usually suffices to give a clear picture of the nature and extent of the condition.

In many cases the twenty-four hour film suffices to make the diagnosis (fig. 1) but if the haustra are unusually deep and the bowel lumen well contracted, segregated masses of barium may give the impression of being caught in diverticula when an opaque enema will reveal that this is not the case. Many diverticula, however, will be missed if the twenty-four hour film above is used, not only because the barium will not fill them, but also because the majority of them are in the descending colon and sigmoid. Because of stasis of the large bowel, it is not unusual to find that the head of the meal at twenty-four hours has just reached the descending colon and there is no barium in the sigmoid or rectum.

By distending the bowel with an opaque enema, diverticula are filled with a fluid medium. In many cases they are not visible because the shadow of the distended bowel obscures the shadows of the diverticula (fig. 2a). After the evacuation of the enema, however, they are well outlined (fig. 2b). Diverticula in the curve of the sigmoid, in common with all other lesions in this area, at times are particularly difficult to visualize. Visibility in this area may be enhanced by rotating the patient to the left as the enema is filling the bowel. Frequently it is much better not to fill the bowel beyond the splenic flexure, because the distal limb of the transverse colon may overlie and obscure the descending colon and sigmoid. When taking the pictures, by rotating the hips to the left or right, diverticula may be brought into view that otherwise cannot be seen.

If diverticulitis is present, nearby diverticula are frequently seen (fig. 3), but in the inflammatory area the usual finding is a constant irregularity associated with some degree of narrowing of the lumen (fig. 4). Complete obstruction to the passage of an enema may be present, although the patient may complain of frequent small liquid stools accompanied by marked pain. The presence of a palpable mass by no means indicates marked encroachment on the bowel lumen. In many such cases the opaque enema

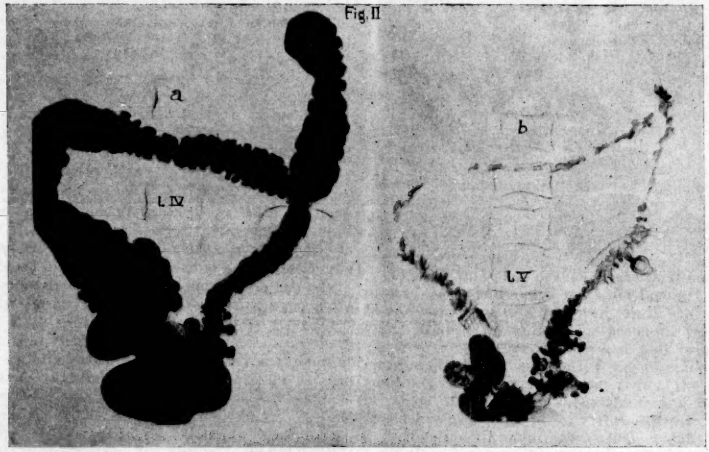

FIG. 2. Diverticulosis:
(a) Opaque enema. Note diverticula in sigmoid.
(b) Same case after evacuation of the enema. Note large diverticulum in descending colon and numerous small diverticula in sigmoid.

seems to pass almost unobstructed, but the affected region is inflexible, the lumen is rigid and some form of irregularity is constant.

The percentage of cases is variously estimated at from five per cent to ten per cent of all cases

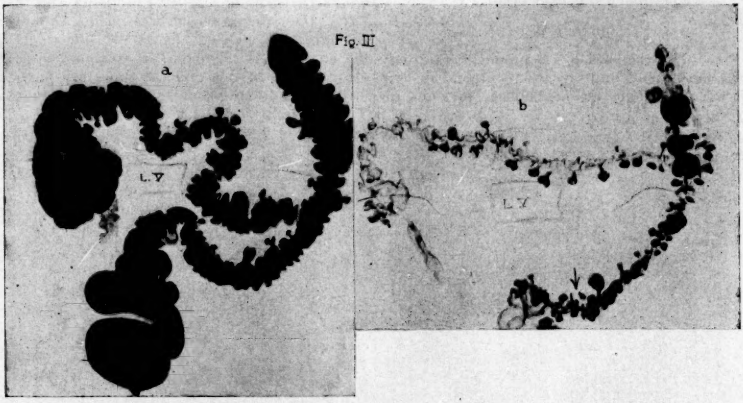

FIG. 3. Diverticulosis and Diverticulitis:
(a) Opaque enema showing diverticula throughout colon.
(b) Same case after evacuation. This case had pain in left lower abdomen, fever, palpable mass. Relieved by medical management, mass subsided.

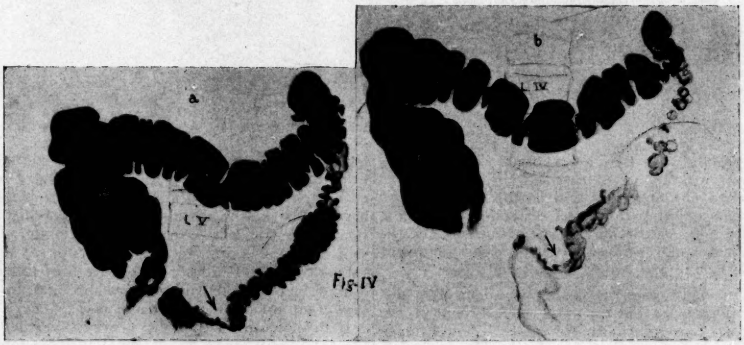

FIG. 4. Diverticulosis and Diverticulitis:
(a) Opaque enema showing rigid, fixed area in sigmoid.
(b) Same case after evacuation. Same rigid, narrow area. Note diverticula in descending colon. (Also had diverticulum of stomach.) Medical management with relief of symptoms.

impossible to visualize after the inflammatory reaction is well advanced. Clinically and roentgenologically the condition may appear to be malignant. Even at operation differentiation is not always possible and the diagnosis can be

having roentgen ray studies. W. J. Mayo[8] has said that twelve per cent of the cases showing diverticula develop diverticulitis. The diagnosis may be suspected by the history and physical findings, but confirmation by the roentgen ray may be difficult. Frequently the diverticula are confirmed only by the pathologic report. A characteristic appearance of diverticulitis, consisting of deep, V-shaped notching of the lumen of the bowel has been described by Spriggs and Marxer[12], but this is not always demonstrable.

TREATMENT

In many articles the treatment of diverticulosis is not mentioned. In others it is dismissed with a caution to keep the bowels open. All writers agree that there is a strong tendency for fecaliths to form in the diverticula, and from irritation by these, associated with a stretched mucosa and a poor blood supply, an inflammatory reaction is apt to start. Where specific treatment is mentioned the chief points advocated are as varied as are the theories on etiology. Some writers advocate keeping the stools liquid. Many are in favor of large doses of mineral oil daily. Bismuth by mouth has strong supporters and draws equally strong condemnation. Strong purgatives once or twice a week have many advocates and few definite opponents. Colonic lavage or irrigations are widely used, the ingredients varying from warm tap water to soap suds, saline, antiseptic solutions or emulsions of mineral oil. The requisite diet is described as being simple, bland, nonirritating, bulky, low residue, or ordinary according to the author's preference.

In our opinion there seems as little justification for giving cases of diverticulosis or mild diverticulitis a strong laxative or purgative as there is for similarly treating a suspected case of appendicitis. The two conditions are essentially similar, both in the pre-inflammatory state and after inflammation has actually set in, except that there is only one appendix and there are usually many diverticula. Even though it be true that many of the diverticula contain fecaliths, it seems unlikely that they can be made to empty or to remain empty by any artificial means. The neck of the diverticulum is almost invariably smaller than the widest diameter of the sac. On the other hand, measures which increase the fluid content of the left side of the bowel are almost certain to force liquid material into the sacs. This liquid material, coming as it does from the right colon without adequate time to become inspissated, is highly toxic, contains many more active bacteria than the normal contents of the left colon, and offers an ideal medium to cause inflammatory reactions in the poorly resistant diverticula.

It seems more logical to attempt to keep the stools formed, to attain one normal evacuation daily, to prevent overactive peristalsis, and to decrease intra-intestinal pressure. These aims are not always to be achieved, but they can be approximated in all cases, and by carrying them out, many threatening cases of diverticulitis can be made to quiet down. These cases all have more or less irritability of the colon as a result of the presence of the diverticula and respond poorly to any form of treatment while they are ambulatory. Bed rest for the initial period of treatment, usually two weeks, is of the greatest value, and cannot successfully be ignored.

A bland, low residue, non-irritating diet, similar to that outlined by Jordan and Kiefer[13] for the treatment of an irritable colon and by Bargen and Victor[14] for the treatment of colitis, is a prime necessity. The diet should have as a basis chiefly carbohydrate foods. At first it should contain no vegetables, no fruits or fruit juices. All fluids and foods should be warm. Obviously such a diet is poor in vitamins, but as the patient's symptoms subside, other elements can be added cautiously. Low residue vegetables are usually tolerated early, and within two weeks cooked fruits can be added. If these additions cause an increase in gas or pain, or if the stools become liquid, they should be dropped promptly.

For the first few days on such a regimen, bowel function is usually poor, but failure of the bowels to move can be ignored because of the low residue character of the diet. Rectal impaction is to be watched for by frequent rectal examinations and prevented by rectal instillation of a few ounces of warm olive oil or corn oil. If the stool has reached the rectum this simple measure usually suffices to cause an adequate evacuation. If the patient complains of an increase in abdominal pain or gas, a small enema (one or two pints) of warm saline solution gives prompt relief.

Of the fruit juices, orange juice taken hot and diluted is usually the least irritating. This should not be given, however, until the movements are well formed and are becoming regular. Tomato juice is poorly tolerated and is better omitted from the menu. All forms of spices and condiments, iced drinks, fresh fruits, the skins or seeds of fruits and vegetables, and coarse bread are also to be strictly forbidden.

Medication by mouth is of little value. Tincture of belladonna, however, seems to alleviate the distress from gas and promote a more natural type of bowel activity. All forms of purgatives, laxatives and even mineral oil should be prohibited.

COMMENT

The foregoing is by no means meant to decry the value of operation in certain cases. It is our feeling, however, that operation should be reserved for the obviously seriously ill cases, those where obstruction is almost complete, where perforation and abscess formation have occurred, or where perforation into another viscus, notably the bladder, causes a complication which demands it.

In the series reported in Table I there were six cases which came to operation out of twenty-one definite cases of diverticulitis. Of these six, three had abscess formation when first seen and eventually died; three recovered. Among the fifteen cases not operated there were two who had large palpable masses which subsided under treatment and a third was almost completely obstructed. This obstruction later disappeared as shown by roentgen ray.

The remaining ninety-six cases of diverticulosis without diverticulitis had treatment similar to that outlined above in addition to treatment for whatever associated diseases were found. No case so treated has developed diverticulitis. All were improved to some degree, and more than half were relieved of all symptoms.

TABLE I

DIVERTICULOSIS AND DIVERTICULITIS, 117 CASES

Diverticulosis—96 cases		Diverticulitis—21 cases	Totals in per cent
Males	33	7	37%
Females	63	14	63%
Average Age	54 yrs.	57.9 yrs.	55.5
Average Weight	158.3 lb.	146.2 lb.	
Other Findings:			
Irritable colon	67	4	61%
Cathartic Abuse	63	11	63%
Duodenal Ulcer	15	1	14%
Achlorhydria	10	1	10%
Cholecystitis	6	2	77%
Diverticula Elsewhere	3	1	3%
	Attacks of pain	21	
	Mass	7	
	Obstruction	8	
	Diverticula visible	14	
	Operation	6	
	Medical treatment	14	
Location of Lesion			
Sigmoid Alone	55	13	58%
Descending Colon and Sigmoid	19	2	18%
Splenic Flexure	2	0	2%
Transverse Colon	10	1	10%
Hepatic Flexure	3	1	3%
Cecum and Ascending Colon	5	1	5%
Whole Colon	2	2	3%
Unknown	0	1	1%
Totals	96	21	100%

SUMMARY

The literature on the etiology, pathology and treatment has been reviewed. A method of treatment of diverticulosis and the milder cases of diverticulitis has been discussed, with special reference to diet. A summary of one hundred and seventeen cases of diverticulosis and diverticulitis is presented.

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ESOPHAGEAL ULCER IN THE NEWLY BORN*

BY MARTIN M. BROWN, M.D.,† AND ELLIS KELLERT, M.D.†

THE occurrence of severe gastro-intestinal hemorrhage in the newly born during the first few days of life with the resultant melena is a startling event before which the attending physician is often helpless. When purpura and abnormal changes in the clotting and bleeding times of the blood are present he may feel secure in the diagnosis of some type of blood defect. Where these symptoms are absent and the bleeding appears to originate from the upper intestine or stomach, the diagnosis may be very difficult, particularly if the possibility of ulcer is not considered. The present tendency is to designate the condition as a form of blood dyscrasia or "idiopathic melena", but careful search in such cases will often reveal a local ulcerative lesion.

Ulcers of the stomach and intestine are not rare in early life. About ten per cent of the numerous cases recorded were found in the newly born. They are usually observed on the posterior wall of the duodenum near the pyloric ring and are thought to be due to digestion of the mucosa following thrombosis. Much more rare,

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apparently, are ulcers of the esophagus, particularly their occurrence in infants prior to the taking of semi-solid food. They are often associated with an infectious process and, as believed by some authors, may follow linear tears of the mucosa due to over-stretching while in an abnormal posture. Kennedy[1], in a discussion of the etiology of duodenal ulcer, states that streptococci or related organisms are the causative agents and that repair takes place so rapidly that ulcers are difficult to find even though the lesion is fairly common. Diamantopoulos[2] believes that ulcer of the esophagus follows engorgement during birth, with the production of fissures in the mucosa and subsequent penetration by bacteria. This author gives an excellent review of the subject up to the year 1926.

We present the following two cases because ulcer of the esophagus is so infrequently demonstrated in the newly born that it is usually not considered by the practitioner when serious gastro-intestinal hemorrhages take place.

CASE I—R. B., male, fifth child, weight 7 lbs. Parents normal; birth normal, no instruments used. Rectal hemorrhages began two days after birth and became increasingly abundant up to the time of death. There was no vomiting. Subcutaneous injections of horse serum, fibrinogen, thromboplastin and father's whole blood were administered without benefit. There was no bleeding from mucous membranes or from the needle punctures. Tests of the clotting and bleeding times were not made. A few purpuric spots were observed about the skin. The infant became increasingly pale and died on the fourth day.

The autopsy was performed eleven hours after death. The only gross pathologic change noted was in the esophagus. A small linear mass of fibrin clot was found attached to the mucosa on the posterior wall of the mid-portion of the esophagus. On removing the fibrin clot, there appeared an area of superficial ulceration measuring 1.5 cm. long in the vertical diameter and 0.4 cm. in the horizontal diameter. The mucosa surrounding this area was slightly injected. The stomach was empty and the gastric mucosa normal. The duodenum contained a considerable amount of blood-stained mucus, and large amounts of blood and mucus were found in the small and large intestines. The intestinal mucosa was normal.

Microscopic examination of the ulceration of the esophagus showed complete loss of epithelium, infiltration with numerous polymorphonuclear and mononuclear leucocytes and the presence of considerable hyalinized material. Mingled with the necrotic surface material were large masses of cocci, apparently staphylococci.

CASE II—Baby R., female, second child, weight at birth 8 lbs. The mother, 26 years of age, and married 8 years, gave a normal medical history. The present delivery was normal after fifteen hours of labor. The father gave a history of prolonged bleeding following tooth extraction and frequent bleeding from the right nostril which, however, was always readily controlled. The baby appeared normal in every respect. The first bowel movements were yellowish-green. The infant nursed poorly at first and on the third day vomited some brownish-yellow material. Bowel movements were normal. On the fourth day the infant cried considerably during the nursing period, and a half hour after the early morning nursing there was projectile vomiting of bright red blood and blood clots. The vomiting of blood was repeated three times during the morning and a large amount of dark-red blood was expelled per rectum. Parent's blood and fibrinogen were administered but pallor became more marked and the respirations rapid. Death occurred five hours after the first appearance of blood in the vomitus. There were no hematologic studies.

The post-mortem examination was performed eight hours after death. Nothing abnormal was found anywhere except in the esophagus. The mucosa of the posterior wall of the esophagus, at a point 4 cm. above the cardiac orifice, contained an area of superficial erosion measuring 1.0 cm. vertically and 0.4 cm. horizontally. There was no thickening or edema about this area but the surrounding mucosa was deeply injected. Fibrin clot was not attached. About 6 cm. below the level of the epiglottis on the posterior wall was a similar but much smaller area of erosion. The stomach was distended by clotted blood, blood-stained fluid and mucus. The mucosa was normal. The small and large intestines contained considerable blood but the mucosa appeared to be normal.

Microscopic examination of the area of ulceration showed disorganized epithelium in the early stages of necrosis. The epithelial layer contained dilated blood spaces. The submucosa appeared thickened and contained dilated blood vessels and interstitial hemorrhage. There was also moderate infiltration of lymphocytes, endothelial leucocytes, plasma and polymorphonuclear cells. The muscle coats appeared normal but the small blood vessels therein were surrounded by many mononuclear cells.

The exciting cause of ulcer of the esophagus in the newly born is unknown. The fundamental process appears to be an injury, infection or both. In older children, injury as a result of swallowing some irritating substance is readily appreciated but such an injury could not take place shortly after birth. Internal trauma in the fetus during the labor period may be more severe than is commonly realized, even after apparently simple and normal delivery. Thus, intracranial and peritoneal hemorrhages are fairly common and occasionally one encounters rupture of the adrenal gland or liver.

Mucosal ulcers of the esophagus have been attributed to tears following extreme torsion and to flexion of the fetus associated with engorgement of the vessels. Marked congestion of the mesenteric vessels and their branches results from sudden closure of the hypogastric artery. Such a vascular change has been cited as the cause of duodenal ulcer. Many experiments are recorded in which the injection of bacteria into the circulation of animals was followed by localization and ulcer formation in the mucosa of the gastro-intestinal tract. Cohnheim observed years ago that necrosis and hemorrhage followed the deposit of bacteria in a vessel wall and this may be the underlying change in esophageal ulcer.

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 19301*

THREE YEARS' INTERMITTENT ABDOMINAL PAIN

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. HENRY D. STEBBINS**: This was a fifty-six year old shoe factory operative of French Canadian parentage. His chief complaint was attacks of abdominal pain of three years' duration.

Three years before entry he began to have non-radiating epigastric pain an hour or two after meals fairly regularly, relieved by soda or vomiting. These attacks would come for a week at a time after every meal and cease for a month, recurring again spontaneously. At first they were not severe enough to inconvenience him or keep him from work. A year before entry they became worse and gradually failed to yield to soda or vomiting except for short periods of time. Six months before admission he noticed that he was becoming very weak and losing weight, eventually totaling thirty pounds. He had several attacks of fainting and weakness during which he sweat considerably. He said after careful questioning that he had dark stools at these times, but he attributed them to powders that the doctor gave him for the postprandial pain. A month before entry his associates noticed that his skin was becoming very pale and of a yellow parchment-like color all over. The pain at that time became constant and would not permit him to sleep. He also had intermittent numbness and tingling in the fingers. Ten days before admission he vomited everything he took. The vomitus was frequently brown. He had several large black stools in the ten days before admission, and became progressively weaker. He had not had any powders for three months.

The past history is unimportant except for an accident to the left eye in childhood with resulting blindness, and the loss of his right leg in a railroad accident sixteen years before entry.

On examination he was well developed but markedly cachectic, lying in bed with obvious weakness but no discomfort. The mucous membranes and the skin were very pale, the sclerae clear. There was no icterus. There was marked dental caries. The blood pressure was 170/100. The radials were palpable, the brachials slightly tortuous. The liver was felt to be down one fingerbreadth below the costal margin, but the edge was never palpated. Rectal examination was negative. The tendon reflexes were normal. The sensation of the fingers was normal.

Laboratory examination showed normal urine. The red blood cell count was 2,000,000, the hemoglobin 45 per cent, the white cell count 7,650. The smear showed 89 per cent polymorphonuclears, slight increase of blood platelets, no marked achromia. The red cells were small. One stool on admission gave a strongly positive guaiac test for occult blood.

The course was uneventful until three days after admission, when it was found that his blood pressure was 110/60. There was no subjective change in his condition however. At 6:30 p.m. he suddenly collapsed and had one of the fainting attacks such as he described in giving his history. The blood pressure was 60 systolic; the diastolic was not obtained. The pulse was 120. He felt weak, sweat profusely and passed a fairly large and soft bloody stool. Transfusion of 600 cubic centimeters of citrated blood was done as soon as possible, whereupon his blood pressure rose to 100/50 and his general condition improved. During the night the blood pressure remained at 110 to 120/60, the pulse at 100, and he slept under morphia. At quarter past six the following morning, five days after admission, he had another attack with collapse and sweating; the blood pressure could not be obtained. Clysis was immediately started and surgeons and a donor were called, but the patient's heart stopped at 6:47, before transfusion could be done.

CLINICAL DISCUSSION

IN DR. CABOT'S CLASS

DR. RICHARD C. CABOT: The pain was relieved by soda or vomiting. We do not so far know whether it was relieved by food.

This history strongly suggests gastric cancer. In the first place it is rare to see gastric ulcer beginning at fifty-three. It does begin then, but we are always surprised. Although many people have it at that age, they have begun to have it at forty or earlier. Then the fact that the trouble goes on to the point where soda and vomiting do not relieve it is much more suggestive of cancer. "Vomitus frequently brown" is what we should expect with cancer, not ulcer; and there seems to be evidence, in the history alone, of anemia, which we expect with cancer and do not expect with ulcer except after hemorrhage. So I expect that as we read on we shall get further evidence of cancer.

"The sensation of the fingers was normal"; we remember that earlier he had had some numbness in his fingers.

"No achromia" is very surprising. I should like very much to verify that statement. It is just the sort of case in which we ought to get

*The following report gives the essentials of two discussions, one in Dr. Cabot's Third Year Class, the other at a conference of the hospital staff.

**Recently senior interne on the East Medical service.

an achromia. It is true that the hemoglobin, if measured correctly, does not show any evidence of achromia.

MISS PAINTER: The record is, "no *marked* achromia."

DR. CABOT: I entirely believe this to be a secondary and not a primary anemia, an anemia wholly dependent on the disease in the stomach.

He had a big intestinal hemorrhage.

In any case which reads straight on, with everything fitting just as it should, one always should particularly ask oneself, what else could this be than the thing which it seems to be? I have seen many wrong diagnoses in cases where people said, "The thing is perfectly obvious." It is in cases where we do not take the pains to set out the various diagnoses that we make the most egregious error.

If the lesion is in the stomach itself, I do not see what else it can be except cancer, because he had a very marked anemia when he got here, and yet he had not had any more hemorrhages so far as we know.

DR. TRACY B. MALLORY: He had had several black stools.

DR. CABOT: Perhaps then I ought to take that back and say that all of his weakness and anemia may be accounted for by hemorrhage, and that it is distinctly unusual to have so many and so large hemorrhages from cancer of the stomach. The condition that usually gives many and large hemorrhages is *ulcer*, and outside of that, *varices in the esophagus* in connection with cirrhosis, splenic anemia and the changes that come with that. His liver may be enlarged. It is doubtful. There is nothing to make us think that he has cirrhosis of the liver. There is no evidence of enlargement of the spleen. The two common extragastric causes of intestinal hemorrhage—cirrhosis of the liver and Banti's disease—I think can be excluded. At any rate we have no positive evidence of them.

This certainly cannot be an aneurysm. Aneurysm does not bleed in this way. It bleeds once for all or in small quantities that do not show. It cannot be a disease of the blood showing itself in hemorrhages in different parts of the body. It cannot be a hemorrhage due to hypertension, nor a hemorrhage associated with leukemia.

I have gone through the items that occur to me, and I see no sense in any of them. I come back to the point where I started, with a diagnosis of cancer of the stomach, though I admit it to be strange that he had so much hemorrhage. It is a somewhat atypical case then, although I can think of no other plausible diagnosis.

Aside from the gastro-intestinal tract we have no reason to think of disease in the other organs. He is an elderly man, doubtless had some arteriosclerosis. I do not think it had anything to do with his death or is of any importance to us.

DR. MAURICE FREMONT-SMITH: Would a non-malignant polyp in the stomach fit it?

DR. CABOT: I think not. I have never heard of a non-malignant polyp leading to so much gastric disturbance. I have never heard of its leading to hemorrhage or to so much indigestion and pain.

A STUDENT: Can he have started with an ulcer that later became cancer?

DR. CABOT: I do not know how we are going to tell about that. My guess is that it is a cancerous ulceration from the start. Supposing it to be cancer, no one of us can ever say what it started from. You cannot say, can you, Dr. Mallory?

DR. MALLORY: Not very often.

DR. CABOT: May we hear what they sent down as the clinical diagnosis?

DR. MALLORY: I think they varied a good deal. I think the final diagnosis was cancer of the stomach, but there were a number of people who thought it was ulcer.

CLINICAL DISCUSSION

AT STAFF CONFERENCE

DR. FREDERICK T. LORD: The diagnosis in this case seemed to rest between peptic ulcer and new growth. As between the two, the indications seem to point rather more clearly to peptic ulcer than to new growth because of the duration and because of the history of massive bleeding, which we associate rather more with ulcer than we do with carcinoma. In addition he had a hypertension when he came in and some arterial sclerosis. He was too ill for us to get the additional facts, such as findings by gastric analysis and x-ray examination, that might have enabled us to make the differential diagnosis.

DR. ARTHUR W. ALLEN: I do not believe I saw this patient.

DR. MALLORY: We must admit that the surgeons were called a little late to be of much use.

DR. ALLEN: I do not believe they would have been of any use in this case if they had been called earlier.

The question as to whether or not a routine procedure might be decided upon for these cases of very severe bleeding from the stomach and duodenum whereby the case might be considered a surgical emergency at any time after the patient's admission to the hospital I think is worth considering, because we have had a certain number of these profuse hemorrhages, particularly from the posterior wall, ulcers in the duodenum that have bled to death before anything could be done for them. Although the condition is one that does not offer a surgeon any great amount of hope that he can successfully operate, it might be better to consider this type of patient as a surgical emergency from the time of his admission into the hospital. If more

of them—there are not very many, fortunately—if more could be operated upon with proper transfusion before they bled to a fatal point, possibly a small number of them could be saved.

It does not seem as though this patient was in any condition for surgery at any time after his admission to the hospital. We had no evidence by previous x-ray examination as to the character of his lesion. He was at an age when he might have been bleeding from an esophageal varix or something else rather than a peptic ulcer. His history certainly would favor peptic ulcer over malignancy. I am in accord with Dr. Lord in thinking that rarely do patients bleed as profusely as this man did from carcinoma.

DR. JAMES H. MEANS: The matter of the amount of bleeding from cancer might be mentioned. I had exactly the same impression, that a massive hemorrhage of this sort comes invariably from ulcer rather than cancer. However, I remember one patient who had just such a type of hemorrhage as this who turned out to have cancer. So that one can find it with cancer, although when it occurs ulcer is far more likely.

DR. LORD: With respect to operation, it must of course be appreciated that these patients present a most difficult therapeutic problem. We are confronted on one hand with the chance that the patient may stop bleeding, pass by this emergency and therefore for the time being recover, and on the other hand he may die; but also under these circumstances operative interference offers a very considerable hazard. I think our tendency up to the present has been rather in favor of trying to get by this emergency with the prospect of resorting to surgery at a more favorable moment. I should like to ask Dr. Allen if he can give us any information to enable us to pick out the cases that might be operated on or that are likely to do better under this waiting policy.

DR. ALLEN: Of course it is not possible to distinguish with certainty a case that will fail to stop bleeding on rest and possibly a small transfusion from a case that will bleed a second or third or fourth time within a short period. The surgical risk is so great that it is not advisable to consider them surgical patients at once. I am sure of that. I think that they should all be given the opportunity to stop bleeding and get into better surgical condition. It seems to me that one might look at it in this way, that if a patient bleeds out his first transfusion in a period of hours rather than of days and has to be transfused a second time, then he probably had best be considered a surgical patient. The only patients who have had two transfusions that have gone on to a better operative risk after a matter of a good many days have been patients who have bled out their first transfusion rather slowly over a matter of five or six days. Patients that have bled out their first transfusion in a matter of hours have always kept on bleeding, and in a good many instances they finally come to the operating room in a very precarious state for surgery. My feeling about it is that if a patient has to be transfused twice in a short time he then should be considered a surgical emergency and an attempt be made to operate regardless of the risk.

DR. CHESTER M. JONES: I should think that immediate transfusion would have been the proper treatment in this case and then if he did not respond, operation.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Bleeding peptic ulcer?
Cancer of the stomach with bleeding?

DR. RICHARD C. CABOT'S DIAGNOSIS

Gastric cancer.

DR. FREDERICK T. LORD'S DIAGNOSIS

Peptic ulcer.

ANATOMIC DIAGNOSES

1. *Primary disease.*
   Multiple duodenal ulcers with erosion of the pancreaticoduodenal artery.
2. *Secondary or terminal lesions.*
   Hemorrhage into the gastro-intestinal tract.
   Pulmonary edema and congestion.
3. *Historic landmarks.*
   Healed localized thrombosis of the pancreaticoduodenal artery.
   Arteriosclerosis, slight aortic and coronary.
   Traumatic amputation of the right leg.
   Absent left eye.

PATHOLOGIC DISCUSSION

DR. MALLORY: The autopsy on this case showed duodenal ulcer. The main surprise, perhaps, was that he had not one but three of them. Two were of considerable importance. They were located just beyond the pylorus, within a centimeter of it, one on the anterolateral surface, the other on the median posterior surface of the duodenum. The one on the anterior surface had become firmly adherent to the gall bladder, as these ulcers sometimes do, had penetrated very deeply, and although it had not actually perforated into the gall bladder, after we began to manipulate it at the autopsy the dividing septum was so thin that we almost immediately ruptured it ourselves. This is the type of case in which a fistula between the gall bladder and the duodenum sometimes develops. Much the more important ulcer was the one on the posterior surface. That one had invaded down into the head of the pancreas and had cut a large branch of the pancreaticoduodenal artery, which was found lying open in the base

of the ulcer with a fresh blood clot loosely attached to it.

Of course whether or not bleeding continues or stops in a case of this sort depends on whether the process of thrombosis in the eroded artery proceeds faster than the erosion. In this case there was rather an interesting finding in that a part of the pancreaticoduodenal artery well back towards the celiac axis was completely thrombosed. The patient had evidently had one or more previous attacks of bleeding, as of course the history tells us. At one of these preceding times extensive thrombosis of the artery had occurred, presumably followed by a long period of remission. Then he developed considerable collateral circulation with more or less complete recanalization of the distal part of the artery, then a second erosion and fresh hemorrhage. The entire gastro-intestinal tract was filled with bright red blood. The final hemorrhage must have been an enormous one, amounting to two liters or more. Of course the death was purely and simply a death from hemorrhage.

DR. LORD: I should like to ask Dr. Allen what his operative procedure would have been.

DR. MALLORY: We have another very similar case. It might be more interesting to discuss the treatment after that has been presented.

DR. ALLEN: The thrombus in this case was probably in the superior pancreaticoduodenal artery and would not necessarily prevent him from bleeding, because this artery anastomoses abundantly with the inferior pancreaticoduodenal and gastroduodenal arteries. That is why it is surgically so difficult to tie the bleeding vessels. You cannot get at them without going under the duodenum.

CASE 19302

A PROBLEM IN TREATMENT OF MASSIVE GASTRO-INTESTINAL HEMORRHAGE

MEDICAL, DERMATOLOGICAL AND SURGICAL DEPARTMENTS

PRESENTATION OF CASE

DR. HENRY D. STEBBINS:* This is the case of a fifty-four year old married Irish fireman who entered the hospital for the ninth and last time through the Emergency Ward with the chief complaint of attacks of abdominal pain for five months previous to entry and extreme weakness for two days.

The long history of reëntries should comprise practically his whole past history. (1) Thirty-six years before the final entry he was in for typhoid fever. (2) Thirty-one years later he was first seen on the Dermatological service for blebs all over the skin. A diagnosis of pemphigus was made. A gastro-intestinal series was done at that time. A normal duodenal cap could at no time be found. (3) He was again admitted on the Dermatological service two months later and stayed seven months. The pemphigus showed an exacerbation. A Graham test was negative. He was transfused, given forty-one doses of ferric cacodylate and improved. (4) He came in again two months after that discharge and stayed six weeks, again with a diagnosis of pemphigus. He was discharged, getting worse in spite of treatment with iron cacodylate. (5) The next admission was fourteen months later. The pemphigus improved with eight injections of tryparsamid. A right direct inguinal hernia began to develop at that time. He was discharged in ten weeks. (6) Three months later he reëntered and stayed two months. The diagnosis was pemphigus. The right inguinal hernia had progressed. Dr. McKittrick advised against operation for the hernia because of the pemphigus. The patient was treated with seven inoculations with smallpox vaccine and four treatments with spleen liquid. There was marked improvement in the pemphigus. (7) He was admitted four months later on the Dermatological service and given three treatments of tryparsamid. He had been very much worse before he came in but was discharged somewhat improved so far as the pemphigus was concerned.

(8) He was next seen on the East Surgical service eighteen months later. The pemphigus was considerably better. He had received twenty doses of tryparsamid in the Outpatient Department and twenty-five doses of ferric cacodylate. He had then been complaining of lower abdominal pain for two months. He was brought into the hospital at that time with the thought that there might be intestinal obstruction from the right inguinal hernia. In the student's history there is a very good description of the abdominal pain which the patient had felt for the preceding two months. It was fairly characteristic pain coming after meals and clearing up with food. A barium enema was done. Operation was advised against and he was given a truss.

Two months after his discharge he was seen in the Outpatient Department on account of increased abdominal pain. Nothing was found on proctoscopy. A gastro-intestinal series was done on the 24th of December. The report had not been obtained until the 29th, when he arrived in the Emergency Ward. He had collapsed on the way to the hospital.

At his ninth and last admission the history of the present illness was of five months of gnawing, aching right upper quadrant, later right lower quadrant and epigastric pain coming on regularly at seven p. m., two hours after supper. He never tried food or soda to relieve it. It would occasionally come on at night. His bowels were constipated. During the five months he vomited two or three times a week with relief of the pain. Two weeks before admission the pain increased and he slept very little. He vomited quite frequently and felt some relief from food or soda. For six nights before admis-

*Recently senior interne on the East Medical service.

sion he vomited every night. At first the vomitus consisted of coffee grounds material. On the last two nights it was definitely black. Four days before admission he thought he had the first black stool, although he said he had not watched the stools before. He had six large tarry stools. The day before admission he was very weak. He fainted twice, once on the day of admission. He had lost thirty pounds in five months.

Physical examination showed a well developed, very pale, very weak man. The mucous membranes were very pale. The back, chest, upper arms and neck were covered with healed and crusted vesicles two or three centimeters in diameter. There were a few fresh vesicles on the forehead and over the cheeks and one or two on the feet. There was marked dental sepsis. The blood pressure was 84/58. The pulse was 100, of good quality, the temperature 97.8° by rectum. There was right inguinal hernia and slight enlargement of the prostate.

The urine was negative. The red blood cell count was 2,500,000, the hemoglobin 30 per cent Tallqvist, the white cell count 10,200, the smear essentially normal.

He came up to the ward and that same afternoon again collapsed. He was not being given any food by mouth and was almost moribund. At six o'clock in the evening he responded immediately to transfusion. On the third day he was still having pain but was apparently in fairly good condition in the interval. The blood pressure was 110, the pulse 100. Again he suddenly collapsed and was almost moribund. He had a second transfusion of 600 cubic centimeters. The red blood count that day was 2,000,000, the hemoglobin 30 per cent, as on his arrival.

On the fourth day he had a third transfusion in the morning because he was still in a precarious condition. That afternoon he had a second collapse out of which he came without transfusion. On the fifth day the red cell count was 1,800,000, the hemoglobin 35 per cent. He collapsed again in the morning and had a tarry bowel movement. He came out of that collapse and returned to his usual state of semiconsciousness, with a blood pressure of 110, without further transfusion. Dr. Allen was called and raised the question whether it might not be a pemphigus lesion which was causing the hemorrhage.

On the sixth day the patient was transferred to the East Surgical service for operation. He was almost moribund at the time he was transferred. Transfusion of 600 cubic centimeters was done before and after operation. He seemed to be in fairly good condition for two days; but two days after operation he developed a slight cough, the respirations rose to 30, and coarse râles were heard, especially at the right base. He was much worse that afternoon. Transfusion of 400 cubic centimeters was done. The chest filled with coarse râles. The temperature did not rise much. On the third day after operation he died.

Clinical Discussion

Dr. Stebbins: At the second admission, five years before the final entry, a diagnosis of duodenal ulcer was made. The report on the gastro-intestinal series done just before his tenth admission was still duodenal ulcer.

At operation a huge ulcer of the duodenum was found and excised.

The discharge diagnoses were bleeding duodenal ulcer, bronchopneumonia, pemphigus and right inguinal hernia.

Dr. George W. Holmes: This x-ray examination was done a year before the last entry. The older films have been destroyed. It shows the characteristic deformity of a large duodenal ulcer with the hyperperistalsis that usually goes with such a lesion. It is interesting that the gall bladder was visible after giving the dye. Some cases of duodenal ulcer fail to show a normal gall bladder shadow; at least that is given as an explanation of the failure of the gall bladder to fill in some cases. In the twenty-four hour film we have a well filled appendix. We think when the appendix is well filled it is probably normal.

Dr. E. Lawrence Oliver: This was from a dermatologic point of view a typical case of pemphigus when the patient first came in. He was in the Skin ward a good many times. He was also in three or four other hospitals. He had a great many ups and downs and gradually improved so far as the pemphigus was concerned, but never got entirely over it. For the last two years the condition changed but little, but at the time of the last acute attack of pemphigus he was rather worse than during the previous year; he had more vesicles than previously. For several years he had had rather the pemphigus foliaceous type, in which the skin peels with a moist surface beneath but with not very many frank bullae.

As to the question of any relationship between bleeding and pemphigus, I think it is very doubtful. The lesions are usually very superficial in the skin. The vesicles and bullae are confined to the epidermis and would hardly be deep enough as a rule to cause bleeding. I think the same is true in the intestinal tract. It is very questionable whether massive hemorrhage could be due to such lesions in the intestinal tract.

Dr. Tracy B. Mallory: When they die what do they usually die from?

Dr. Oliver: Pneumonia or intercurrent infection rather than the pemphigus itself. This disease should not be considered fatal. It lasts a long time. Sometimes it clears up entirely and may stay well for any number of years before another attack. In one very interesting case, a woman at the age of twenty,—this was sixty years ago,—Dr. James White made a positive

diagnosis of pemphigus. The disease cleared up entirely in about a year. Twenty years later, at the age of forty, she had another attack. Dr. John T. Bowen took care of her and made a diagnosis of pemphigus. That attack cleared up in a year. She had a third attack when she was more than eighty years of age. During that attack she died after suffering a year or so. That shows how extraordinary these cases are and what long periods of remission they may occasionally have. As a rule however they do not have such remissions.

DR. MALLORY: This is the second case of pemphigus that has been associated with duodenal ulcer in the past few months. I imagine that is merely a coincidence.

DR. CHESTER M. JONES: It seems to me that this case illustrates in the first place that at times here in the hospital in cases with several diagnoses one of them not infrequently drops out of sight. In this case the ulcer was not taken seriously for a long period of time. As far as the emergency is concerned they have underestimated the danger of waiting indefinitely, as was done here. It would seem to me that at the time he came in, practically in shock, the question of surgical intervention should have been considered.

The other thing which it seems to me this case brings out is that an operation was performed, and apparently a successful one, on a patient dying of something else. If it had been possible to operate on him thirty-six or forty-eight hours before, there is reason to believe that he might have been saved. Operation was done under unfavorable circumstances, due apparently to the fact that they waited too long. I think this group of bleeding ulcers is a very interesting group. There is a good proportion of them that will recover from at least their initial hemorrhage perfectly well. There is a border line group in which it takes very nice judgment to determine the wisest course. I think it would be well worth while to have these patients seen from the point of view of surgery on entry and a decision made at once as to what should be the steps undertaken in the next twelve hours.

DR. FREDERICK T. LORD: With respect to what Dr. Jones has said, the problem made a somewhat different impression on me. The patient was in a desperate condition from the time he entered the hospital until he was seen by the surgeons four days later. It seemed to me that during this time he was too ill to be subjected to an operation of such magnitude without giving him the opportunity to rally. I realized the risk from hemorrhage, but thought it best to wait and only reluctantly consented to operation when he failed to rally.

DR. JONES: The point I should like to make is this. He was in the Medical ward I believe about three or four days, though he was in a very critical condition, practically in shock, and the result of transfusion was not satisfactory. It seems to me no matter what the risk is one should make an attempt to stop the bleeding then and there. We are going to lose a number of these patients unavoidably by operation, but regardless of that risk I think it ought to be assumed rather than to wait hoping that the patient will rally. It is very hard to judge beforehand and say this is a clean cut group. I do not believe it is a clean cut group. I think it is always an individual problem. In some of these cases we should transfuse at once and watch for results. If the results are not satisfactory go right ahead with a second transfusion and perform whatever operation can be done. The large hemorrhages almost always involve the pancreaticoduodenal artery, and as a rule a good coagulum will not form, and the hemorrhage will be a fatal one.

DR. ARTHUR W. ALLEN: Unfortunately a certain number of these severe bleeding ulcers are brought to the hospital by their first massive hemorrhage. It has been repeatedly stated in the literature that people practically always recover after their first attack of bleeding. We have had quite a number of these severe cases which although they may have bled in small quantities before, never have bled sufficiently to bring them to the hospital. We are bound to confront a problem in this group.

Of course in the group that stop after the first severe hemorrhage, if operation is not done in a quiescent state we have room for criticism among ourselves and from the patients' point of view, affecting their willingness to be operated upon. But we must be prepared to meet this group of initial massive hemorrhages, usually occurring in people with some arteriosclerosis. Where the hemorrhage is so profuse, so sudden, and comes again very shortly after transfusion, we must assume that we are dealing with an open vessel of considerable size, and we ought to develop a technic whereby these patients can be operated upon as soon as we can be convinced that they are likely to continue to bleed. The greatest help in this group would be an x-ray that had been made some time previously to show us that the ulcer was on the posterior wall of the duodenum. If we have that as evidence, then we can usually eliminate the other causes of fatal bleeding, such as bleeding from a severe gastritis or an esophageal varix. So in those with previous x-ray evidence we can go ahead with a good deal of assurance that we shall find an ulcer that has gone through the wall of the duodenum and has eroded the pancreas down to the gastroduodenal or pancreaticoduodenal artery.

The only satisfactory way to operate in that region is to lift up the distal end of the stomach and get at the ulcer area from behind. It has been pointed out by Dr. Daniel Jones that probably that is the only way we can successfully operate on these patients,—do a transection of the stomach and remove the distal segment with the ulcer bearing portion of the duodenum. That has been tried here unsuccessfully

in the past. Opening the duodenum over the ulcer and attempting to pass ligatures through and around the ulcer has also been tried, as well as simple gastro-enterostomy, both unsuccessfully. What has happened usually is that they have bled severely during the operation. So it occurred to me that a modification of the transection of the stomach and a lifting up of the posterior wall, getting at the ulcer from behind, might be worth trying. This modification consists in quickly cutting across the stomach at the junction of the lower and middle thirds, detaching the lower clamp and opening the distal portion of the stomach anteriorly through the pylorus and the first portion of the duodenum, exposing the whole region quickly. Then one can deal with the hemorrhage temporarily by placing a finger over the opening in the artery. Resection of that portion of the stomach and duodenum can then be done deliberately. That is what we did with this patient. This man's condition as far as blood pressure is concerned improved during operation. It was very spectacular, for we usually see them fade out during the procedure. He had the largest erosion into the pancreas that I have ever seen, measuring 2.5 to 3 centimeters in diameter. You realize what great difficulty there might have been in getting sufficient duodenum above the ampulla of Vater to be safe in turning in the blind end of the duodenum. One has no duodenum on the posterior wall in the bed of the ulcer. The chronic process of scar tissue formation that has gone on in this region will have foreshortened the area between the duodenum and the liver so that one is likely to injure the common duct or hepatic artery in the closure of the duodenum. In this case, although we did not have much room to spare we felt that we were definitely above the ampulla at the level of turning in the duodenum.

The reason we drained him was that we felt he was due to get a certain amount of oozing from the ulcer area in the pancreas and thought that perhaps we could drain that region out through his flank and prevent the pancreatic juice from affecting the sutures. This man was living on borrowed blood at the time of operation, and we know that a man can live on only a certain percentage of alien blood. If he has too many transfusions spaced close together he will die no matter what we do. We do not know how long it took the ulcer to form; but in order to get at it at a favorable time we probably should have had the opportunity before he came to the hospital. Of course we had no way to do that. It might be said that if he had been operated upon twenty-four hours after admission the outcome would have been different. I am inclined to think it would not have been in this case. I think that most of the ulcers in this location would not be so large, would not involve so much of the pancreas or cause so much danger of peritonitis if we could get them in the early stages of severe massive hemorrhage.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Bleeding duodenal ulcer.
Bronchial pneumonia.

ANATOMIC DIAGNOSES

Bleeding duodenal ulcer.
Operative wound, posterior Polya.
Subdiaphragmatic abscess.
Bronchopneumonia.
Pulmonary collapse.
Hydrothorax, right.
Arteriosclerosis, slight, aortic.
(Pemphigus).

PATHOLOGIC DISCUSSION

DR. MALLORY: At autopsy we found a subdiaphragmatic abscess and a pleurisy on the right side above the abscess. The infectious process had obviously extended directly through the diaphragm. I think that was the immediate cause of death.

The operative wound itself was as Dr. Allen has described it. The end of the duodenum had been turned in and there was just a centimeter leeway between it and the ampulla of Vater, but it was safely above it. There was no sign of leakage from any of the suture lines. The infection of the subdiaphragmatic space must have occurred from the bed of the ulcer, which could not be entirely sterilized. That is a risk that must inevitably be taken in any case of this sort.

DR. JONES: It seems to me that there is one other thing that might be said on this subject of bleeding ulcer. Both of these patients were over fifty, and I judge that this patient was not particularly well nourished. In the younger group of well developed and well nourished people I think it is probably true that most of them will get through hemorrhage perfectly well and recover. In the older group the patients do not do so well. There is a recent note by Dr. Minot that may have some bearing. Where there is undernutrition of any degree there is distinctly faulty coagulation and there may be a diminished tendency to clot formation and a greater tendency to prolonged bleeding. In the older group some other disease or age itself may complicate the picture. I think these groups can be subdivided into a younger and well nourished group and the older group, patients who come in pretty bad condition and have massive hemorrhage.

DR. ALLEN: Were there not a great many areas of fat necrosis?

DR. MALLORY: Yes, a moderate number in the region of the head of the pancreas and the exposed bed of the ulcer, although I do not think that was very significant in comparison with the subdiaphragmatic abscess.

DR. BETH VINCENT: Most surgeons, I think, agree that duodenal ulcer is a case for medical treatment until that form of treatment, after thorough trial, has proved a failure, or unless there occurs obstruction or perforation. I should

like to raise the question whether excessive hemorrhage does not also indicate an ulcer case that should be under the care of a surgeon. By excessive hemorrhage we mean bleeding so profuse or persistent that the patient's life is endangered by the loss of blood. I should like to ask the medical men if in their experience massive hemorrhage does not usually indicate a type of ulcer that resists medical treatment. If it is true that they cannot hope to be successful with medical treatment alone, should they not consult a surgeon at once, as Dr. Jones suggests, and let him select the time and type of operative procedure?

Dr. Lord, do you remember any case with massive bleeding in which the patient survived and the symptoms were controlled by medical treatment?

DR. LORD: We see all degrees of severity in the way of bleeding from peptic ulcer. In general, a large proportion of patients recover from the hemorrhage. If we restrict the discussion to those with excessive bleeding comparable to the problem under discussion, I can refer to two patients who died and three who recovered. Of the latter the following is the most severe example: In the case of a woman of forty-five there was the history of bleeding three years before she entered the hospital for a recurrence beginning two days before admission. The bleeding lasted fifteen days during which the red count fell to 990,080 and the blood pressure, though not persistently low, was on one occasion 70/0. Two transfusions were given. The x-ray findings were consistent with duodenal ulcer. The patient recovered without operation. Such experiences illustrate the difficulty of estimating the outlook and raise the question of the desirability of resorting at once to a major operation on patients already desperately ill from hemorrhage. To do so is to incur the additional hazard of shock and postoperative complications and a very high operative mortality, and it may well be a question whether the patient might not have survived without operation. I should like to ask Dr. Jones his opinion.

DR. JONES: I think the patients whom we have lost from hemorrhage here have been the ones who in the first twenty-four hours have had nothing but expectant treatment. They do not rally as they should. If they have not been transfused it is a mistake, or if they were transfused they have not responded so well as they should. This particular patient had had tarry stools for three days before admission. In other words he had had quite a profuse hemorrhage for several days and then came in in shock with a hemoglobin of 30 per cent, which is a very critical level. When you have as low a hemoglobin as that in a patient with such bleeding and then transfuse and the patient goes into shock again and has another hemorrhage, I think that is a definite criterion for operation. In other words, if the patient starts at a very low level and in spite of immediate transfusion bleeds again, I think that is a perfectly good reason for assuming that that patient will probably not do well. If he has no further hemorrhage after transfusion and the pulse and blood pressure come back to normal, then I think there is perfectly good reason for waiting another day or so, and I think it is a question of a decision every few hours.

This man is an extreme example and I think the group is a small group out of the total number that have bleeding of any degree from ulcer, but it should for various reasons form a fairly consistent picture. Is not that your impression, Dr. Allen? They have come in in pretty bad shape and do not respond satisfactorily.

DR. ALLEN: I should say that is true. There are undoubtedly a great many cases of massive hemorrhage from duodenal ulcer that will stop and get into a comparatively normal state of health. I am not sure that that answers Dr. Vincent's question. We have in our records of bleeding ulcers a great many cases that recovered. I think only seventeen were fatal out of eighty-odd cases that we looked up. Is that right, Miss Stockbridge?

MISS MAUDE M. STOCKBRIDGE: There were eighty-three cases of severe massive hemorrhage in our first group.

DR. ALLEN: There were seventeen fatal ones. The rest of them got over that particular hemorrhage. Of course every one of those patients who recovered should have been subjected to surgery probably, because a certain number of them are going to bleed to death at second or third hemorrhages.

DR. JONES: I think the answer is that these patients with severe gastro-intestinal hemorrhage from probable ulcer should constitute a group that demands immediate consideration as to operation within six or twelve hours of their admission.

In answer to Dr. Vincent's question, I think there is no doubt a fairly large group of ulcers, regardless of whether they bleed or not, which if they have been followed for a reasonable length of time should be considered as cases for surgical intervention; at least they should be discussed from that point of view. I do not think the medical men should take the entire responsibility of handling the cases. I think that in the group of massive hemorrhage one should always consider the question whether surgery is advisable even between the attacks rather than wait until the patient gets into a critical condition.

DR. ALLEN: Jordan and Kiefer have reported a series of uncured cases of duodenal ulcer treated by medical régime. They found that bleeding headed the list of symptoms that place an ulcer in the group of those that do not respond satisfactorily to medical treatment.

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EXPERT MEDICAL OPINION

ONE of the problems common to medicine and law, persistent because of the nature of humanity and recurrent in forcing itself into the focus of attention because no adequate procedure has been devised for its solution, is that presented by expert medical testimony in court. At times occasion for scandal to one or both professions, it represents, nevertheless, a necessary resource in the struggle for justice. It would seem to be capable of filling a high place as honorable as the degraded position in which it is sometimes found, is dishonorable. Who is at fault and where does the remedy lie?

In the legal procedure the responsibility lies first with the bench and the bar, for the attorney brings in the alleged expert and the court accepts him. But is the court itself in a position to pass on the qualifications of a medical expert? Is not the court called upon to perform a function for which it may not have the qualification? At any rate the court sometimes fails to perform this function well.

Recently in a court in Massachusetts, there testified as an expert in obstetrics a man who had practiced for many years. Under cross-examination he was vague about the amount of obstetrical work he had done in the previous twenty years. Naturally he could not remember how many patients he had attended. There was a pertinent fact not brought out in court, a "stubborn and irreducible fact", that during the twenty years previous to his testimony there had been paid to him by the large city in which he had been practicing not one twenty-five cent fee for filing a birth return. Yet he was introduced by counsel as an expert in obstetrics and was accepted by the court.

It has been stated publicly that it is impossible in Massachusetts to get a physician to testify against another physician. This is the interpretation of the situation by an advocate and illustrates the difference in the points of view of the attorney and of the physician. It should not be difficult to find physicians who will go into court and testify as to generally accepted views on a medical question at issue, or to give their own opinions on the question. But the advocate, the attorney does not want this; he would regard himself as unworthy of the confidence of his client if he did not use every legal means to exclude truthful testimony which he judged likely to impair his chances of getting a verdict for his client. In court, he is interested in bringing out only the facts that will impress the jury in favor of his client; also he is interested in suppressing in every legal way facts which might tell against his client. If such facts cannot be suppressed, he tries to distract attention by minimizing their weight, re-interpreting them, confusing the witness or discrediting him if possible.

The physician in his practice, on the other hand, welcomes testimony from any and every source if it throws light on the case. The physician seeks all the truth. The advocate seeks only what he can use to his own advantage.

The uninitiated, to whom the "rules of evidence" of court procedure are truly esoteric, thinks of them as devised to assist the defendant in some mysterious way. He does not know they were developed to protect the jury from the advocates, as is indicated by the relaxations in hearings without jury.

It is not strange, therefore, that the physician, rarely a match for the attorney in making the worse appear the better cause, shuns counsel for both plaintiff and defendant, and especially when his testimony is sought against his "own brothers", to use the words of the Hippocratic oath.

But physicians do make mistakes, and there are some physicians who do malpractice; and in securing justice the court must have expert medical opinion. So here is a problem that concerns the public welfare, and since it is certain there are men of *good* will in both the legal

and the medical professions, the question is, Are there not men in both professions who also have *will* enough to meet together and work out on common ground a practicable solution of this problem? The fields are white already to harvest.

Singularly we have in this issue two letters bearing on court procedures. Theoretically the courts are created to administer justice but it is not always possible to secure this. The fact that considerable attention has been paid to the possible solution of the difficulties important to the parties in interest and the two professions concerned, would seem to make it advisable to carry through the suggestion of having the medical and legal professions take constructive action. It is now squarely before the medical profession and it is eminently desirable that there should be an effort to find a solution of the problems involved. It would be most unfortunate if the medical profession refrained from taking the initiative. A committee representative of the best opinion of physicians should be selected to take this matter under advisement and arrange for conferences with the leading members of the Bar with the hope that some constructive action may follow.

A THIRTEEN MILLION RECOMMENDATION

Dr. James V. May, Commissioner of Mental Diseases in a hearing June 27 before a Committee of the Massachusetts Legislature and the municipal finance commission sitting jointly presented a hospital construction program calling for expenditures of thirteen million dollars to care for the mentally ill. Dr. May explained that by this expenditure accommodations could be provided for 4683 additional patients, that there is a large waiting list for institutional care and that, together with future demands, necessitates these additions to the several institutions as follows: Metropolitan State Hospital, Waltham, $1,550,000; Boston State Hospital, $2,330,500; Danvers, $325,500; Foxboro, $255,000; Grafton, $650,000; Medfield, $155,000; Northampton, $1,734,000; Taunton, $1,375,000; Westboro, $755,000; Worcester, $93,000; Gardner State Colony, $100,000; Belchertown State School, $1,481,500; Walter E. Fernald State School, $946,000 and Wrentham State School, $489,500.

In addition an outlay of $400,000 is recommended for a psychological research institute at the Boston State Hospital. It is disconcerting to find in the report that adequate fire protection which will cost more than $5,000,000 is needed in addition to the sums stated above.

The possibility of the dire prediction of Dr. May seems to demand immediate action for it may be that it would be a waste of money to provide the fire protection without the other appropriation and if Massachusetts should incinerate scores of its public charges a blot would be placed on her fair reputation.

There should be no delay in the creation of a psychological research institute, for present conditions demand intensive study of the causes leading to mental illness. Better and more general preventive measures will be a good investment for the State.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Warren, Shields. A.B., M.D. Harvard University Medical School 1923. Pathologist to the New England Deaconess, New England Baptist, Collis P. Huntington Memorial and Pondville State Hospitals. Consulting Pathologist, House of the Good Samaritan. Instructor in Pathology, Harvard University Medical School. His subject is "Studies on Tumor Metastasis. 3. The Distribution of Metastases in Carcinoma of the Large Intestine." Page 167. Address: 195 Pilgrim Road, Boston.

Heath, Clark W. A.B., M.D. Harvard University Medical School 1926. Assistant in Medicine, Harvard University Medical School. Assistant Physician, Thorndike Memorial Laboratory, Boston City Hospital. September, 1932 to June, 1933 was spent in Medizinische Universitätsklinik, Prof. Morawitz, Leipzig, Germany. His subject is "The Volume and Hemoglobin Content of the Red Blood Corpuscles in the Light of Recent Knowledge of Anemia." Page 173. Address: Thorndike Memorial Laboratory, Boston City Hospital, Boston.

Swan, Channing S. A.B., M.D. Harvard University Medical School 1923. F.A.C.S. Assistant Urologist, Massachusetts General Hospital. His subject is "Perinephric Abscess." Page 180. Address: 264 Beacon Street, Boston.

Dufault, Paul. A.B., M.D. Montreal University 1924. Assistant Superintendent, State Sanatorium, Rutland, Mass. His subject is "Prolonged Bed Rest Treatment in Pulmonary Tuberculosis." Page 184. Address: State Sanatorium, Rutland, Massachusetts.

Curran, John F. M.D. Tufts College Medical School 1912. F.A.C.S. Junior Surgeon, East Surgical Service, Worcester City Hospital. District Surgeon, New England Telephone and Telegraph Company. Address: City Hospital, Worcester, Massachusetts. Associated with him is

Goodale, Raymond H. B.S., M.D. Harvard University Medical School 1924. Pathologist, Worcester City Hospital, and Harrington Me-

morial Hospital, Southbridge, Massachusetts. Instructor in Parasitology, Boston University School of Medicine. Address: 55 South Lenox Street, Worcester, Massachusetts. Their subject is "Ectopic Pregnancy." Page 189.

AYMAN, DAVID. A.B., M.D. Harvard University Medical School 1921. Assistant in Medicine, Beth Israel Hospital. His subject is "The Hereditary Aspect of Arteriolar (Essential) Hypertension." Page 194. Address: 371 Commonwealth Avenue, Boston.

WILKINSON, S. ALLEN. A.B., M.D. University of Pennsylvania 1925. Department of Gastro-Enterology, Lahey Clinic, Boston. His subject is "Diverticulosis and Diverticulitis of the Colon." Page 197. Address: 605 Commonwealth Avenue, Boston.

BROWN, MARTIN M. M.D. Rush Medical College 1888. F.A.C.S. Attending Surgeon, North Adams Hospital. President, North Adams Hospital Staff. Address: 117 Main Street, North Adams, Massachusetts. Associated with him is KELLERT, ELLIS. M.D. Albany Medical College 1909. Pathologist, North Adams Hospital, Putnam Memorial, Plunkett Memorial, Ellis Hospital, and Eastern New York Orthopedic Hospital. Address: Ellis Hospital, Schenectady, New York. Their subject is "Esophageal Ulcer in the Newly Born." Page 202.

MISCELLANY

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

HIGH BLOOD PRESSURE*

BY LAURENCE B. ELLIS, M.D.

Patients are constantly asking doctors whether they have "blood pressure". Of course they have; everyone alive has a certain blood pressure. What these people want to find out is whether their blood pressure is unduly elevated. So much has been said and written about "high blood pressure" or hypertension, that the importance of this condition is well recognized by the laity but at the same time many misconceptions regarding it are prevalent.

It *is* a very important condition to know about because it is exceedingly common in people of middle age and over and, in its late stages, is responsible for a great deal of heart and kidney disease and apoplexy. I propose today to tell you first something of what is known about the nature and cause of this disease, "high blood pressure," and second a little about the general measures to take in its prevention and treatment.

*May 2, 1933, Station WBZ, 4:30 P.M.

What do we mean by "high blood pressure"? Let me make a comparison. The human blood vessels are very similar to any system of pipes, such as the water supply system to your community. There is a central pumping station, the heart, that sends the blood out through the large arteries which are like the great water mains. These gradually split up into smaller and smaller vessels or pipes until finally the smallest vessels—the capillaries—are reached where the necessary and important gases and food stuffs the blood has been carrying are given off to the tissues and the waste materials are taken aboard. The return journey of the blood to the heart and lungs takes place by way of the veins.

As the blood is pumped out by the heart a certain pressure is normally created in the arteries, just as there is a pressure in the water pipes leading to the faucets in your sink. This pressure in the arteries becomes less and less the farther the blood gets from the heart so that by the time the capillaries are reached the pressure is practically nothing at all.

What happens when high blood pressure first begins is that the smallest arteries become much narrowed due to a spasm of the vessels. As a result the heart has a much harder time pumping the blood through these narrowed vessels and so the pressure in them goes up. For a time this may do no harm, sometimes for many, many years, but in most cases this excessive pressure sooner or later causes weakening and changes in the walls of the blood vessels, or the heart may become exhausted from its constant task in maintaining the high pressure. So you see, that high blood pressure is a condition which affects the whole body of the person who has it. It is not confined to the kidneys or the heart or the brain.

Next, what causes this high blood pressure? I am afraid that I shall have to admit at once that no one knows the exact cause of this condition, but we do know a good many facts about it that help us to understand why some people have it and others do not.

There is no doubt that high blood pressure is much more prevalent in some families than in others. Perhaps in some of these cases there is an actual inheritance of a weakness of the blood vessels. But more commonly, probably, it is an inheritance of a general type of personality. People that tend to worry a great deal, that are always keyed up and under tension seem to be particularly prone to develop this condition. This is especially true when individuals of this type find their lives cast in rôles where the conditions leading to mental tenseness are marked. So we see it developing in business men who drive themselves in an exacting business; we see it in persons with an unfortunate environment, such as an unhappy home life.

There are other factors which seem to play a

part in bringing on high blood pressure in some people. Obesity is one of these; and it is just one more reason why fat people are poor insurance risks. Think of all the people over seventy you know. How many are fat?

What you people are really interested in is not what causes high blood pressure but what you can do to keep from getting it; or if you already suffer from it, what you can do to prevent it from getting worse.

Like so many chronic diseases, it is probably easier to ward off high blood pressure or to treat it in its early stage than to accomplish much after it is far advanced. The time to treat diseases successfully is when they are just beginning; before they give rise to symptoms. That is why periodic health examinations are so important. Have your doctor check up your physical condition at least once a year, and you may avoid all sorts of trouble and expense in the future. Of course, if you already have high blood pressure, you should be under the care and supervision of a reputable physician who will advise you as to what special measures and medicines you should take.

Far more important, however, than is any medicine, in the treatment of high blood pressure is the mode of life one lives. A few general words of advice as to this should prove of value to those who have the disease and to those who wish to do what they can to avoid it.

The essence of this advice is to live moderately, avoid excesses, and learn to relax both physically and mentally. A sad commentary on the type of living that American civilization breeds is the fact that there is far more high blood pressure in this country than elsewhere. We are a people given to excesses, to constantly driving ourselves in our work and our play, and few of us really know how to spend our leisure, how to relax.

A habit of relaxing must be acquired. Life must be taken philosophically; there must be reasonable rest periods during the days, plenty of sleep at night and proper vacations. Recreation and play are important but they should be taken in the spirit of fun and not as perpetual contests. All our habits of living should be moderate. Excessive eating, drinking, smoking are all unwise. These are hard habits to cultivate in people who are used to the opposite mode of life, but acquired they must be, and they will frequently pay generous returns in greater health and prolonged life.

THE MORTALITY RATE OF DIABETES

According to Dr. F. L. Hoffman, as quoted in the *New York Times* of July 21, the diabetes mortality during 1932 reached the highest rate on record, that of the United States exceeding that of any of the representative nations. Data collected from fifty American cities since 1912 indicate a rise from 15.9 per 100,000 persons in 1912 to 26.3 in 1932. In the United States registration area the rate was 20.4 in 1931. The figures from some other countries are 14.5 for England and Wales, 14 for Scotland, 13.9 for Australia, and Northern Ireland 9.6. Iceland has a rate of 0.9 and Siam 0.4.

It is suggested in the report by Hoffman that the mortality rates seem to coincide with the proportionate amount of sugar consumed in the several countries. It is well known that the per capita consumption of sugar in the United States is very high.

MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending July 1, indicate a mortality rate of 10.2 as against a rate of 10.1 for the corresponding week of last year. The highest rate (18.3) appears for San Antonio, Tex., and the lowest (5.6) for Akron, O., and Flint, Mich. The highest infant mortality rate (10.0) appears for Atlanta, Ga., and Bridgeport, Conn., and the lowest for Cambridge, Mass., Canton, O., Duluth, Minn., Evansville, Ind., Fall River, Mass., Kansas City, Kans., Long Beach, Calif., Portland, Ore., Salt Lake City, Utah, Schenectady, N. Y., Seattle, Wash., Spokane, Wash., Tacoma, Wash., Utica, N. Y., Wilmington, Del., and Youngstown, O., which reported no infant mortality.

The annual rate for 85 cities is 11.5 for the twenty-six weeks of 1933, as against a rate of 12.0 for the corresponding period of the previous year. June shows a slight increase of the figures of the corresponding month of last year. — *Bureau of the Census.*

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OFFICERS ELECTED BY THE BOARD OF REGISTRATION IN MEDICINE FOR THE ENSUING YEAR

The Commonwealth of Massachusetts
Department of Civil Service and Registration
Board of Registration in Medicine
State House, Boston

July 14, 1933.

Editor, *New England Journal of Medicine,*

At the annual meeting of the Board of Registration in Medicine, held on July 13, 1933, Dr. Charles P. Sylvester was reëlected Chairman, and Dr. Stephen Rushmore was reëlected Secretary, for the ensuing year. Dr. Edward A. Knowlton was designated to act with the Chairman and the Secretary in the examination of chiropodists.

Very truly yours,

STEPHEN RUSHMORE, M.D., *Secretary.*

A YELLOW FEVER OUTBREAK TURNED TO ACCOUNT: YANKEE INGENUITY IN THE SOUTH

Editor, *New England Journal of Medicine,*

The following story is from "Retrospections of America," 1777-1811, by John Bernard, some time Secretary of the Beefsteak Club, and Author of "Retrospections of the Stage." (New York, Harper and Brothers, 1887).

Bernard was a well-known English actor, who came to America to seek fortune in the new country, from his profession, and pleased many audiences. His book will prove fascinating reading for physicians who are not familiar with its Dickensian accounts of our early times.

The Story:

"I was told a story of a 'Down Easter' mode of creating a demand for a supply, which amid all the ingenuities of modern commerce may fairly claim originality. One of the class called a 'hickory dealer,' or seller of wooden ware, came down to the south in summer-time with a well-laden wagon, but was destined to encounter a sweeping opposition in the Yellow Fever, which had commenced business about a week previous in such a wholesale way that the only wooden ware in requisition was a coffin.

"The ravages of the plague were at this time so dreadful that it will be supposed there was a genuine tendency to try the most desperate and absurd expedients to avert it, though many such proved but pioneers to its progress. This the pedler was aware of, so resolved, as regarded his own fortunes, to extract good from evil. Dressing himself as respectably as possible, he mounted his horse and rode up to a printing office in Williamsburgh, when, under an assumed name, he had a hundred bills struck off to this effect:

'WANTED IMMEDIATELY

'Wooden ware in any quantity for the fever hospital in Philadelphia, such being found not to convey the infection.

By order of the Board of Health,

SETH ADAMS.

'N. B. All persons are cautioned how they use crockery, which is the cause of plague to thousands.'

"Fifty of these placards he sent his boy to stick round the streets of a village lying in his road, when the fever had begun to show itself. A discovery so important, so simple, and apparently in such close connection with a remedy, created an instant sensation.

"The doom of crockery was pronounced. Jugs, bowls, basins, teapots, and other utensils most esteemed or necessary were hurled out of the windows, in showers. No grandmother's gift, no ancestor's relic, survived this fall of China; the streets soon exhibited a series of domestic tumult, and looked like a pottery after an earthquake.

"About noon, when the work of destruction was at its height, a wagon made its way into the village, with a man vociferating with all the power of his lungs, 'Wooden Ware!'

"His arrival was hailed as a God-send; a crowd collected round him as to a magician who brought talismans; and in less than two hours his plague averting platters were all disposed of at exorbitant prices." Very truly yours,

Prouts Neck, Maine, WM. PEARCE COUES, M.D.
July 14, 1933.

A CRITICISM AND A REMEDY

July 7, 1933.

Editor, *New England Journal of Medicine,*

Of late there has been somewhat widespread criticism of the attitude of the medical profession toward malpractice actions, criticism originating, to be sure, in legal circles, but spreading.

In spite of the very small proportion of suits of this sort that have merit, we must admit, at least, that there *might* be a justifiable suit of this nature and that in such a case there would be real difficulty in presenting high grade medical testimony in its support.

This is only natural and it is impossible to condemn anyone for not wanting to mix up with the run of these cases.

At the same time, the present situation is unsatisfactory, not only from this point of view, but in that the presentation of the *defendant's* testimony is often under present procedure very unsatisfactory.

In cross-examination which is often absolutely unessential, half-understood technical details are apt to occupy the foreground.

Only too often the jury gets a poor chance to form any sort of clear picture of the actual issues involved.

It has seemed to me that there might be relief from this situation.

Do you regard as practicable the plan here proposed of a tribunal of medical men to pass on each case and report to the court on the technical medical aspects of the case for the information of the court?

There is a precedent in the form of a provision for examinations as to sanity in criminal cases in this state—a provision which seems to have been productive of unmixed good and of which I, for one, have heard no criticism.

If a procedure of similar scope, not taking the place of a trial, not depriving anyone of his traditional right to his day in court, could be worked out for malpractice cases, it might similarly help in clarifying the situation in that field.

It would be possible, I think practicable, to have a list of medical men, acceptable to everyone (as the psychological experts seem to be), men of recognized standing and fairness, appointed by the Governor, constituting a limited

board, not less than two of whom might be assigned to hear and report on each case of alleged malpractice.

An arrangement whereby the plaintiff's attorney should present the outline of his case to the board representatives, before bringing a suit, would help. This arrangement might be either permissive or obligatory.

Of course it is recognized that anything of this sort would mean much working up of details. That is true of anything worth while and it would probably be perfectly possible to put this into working form.

The real question is whether this suggestion contains enough promise of relief to commend itself to the profession, to the courts, and to the people.

Signed,
FREDERIC JAY COTTON.

WHAT CAN BE DONE ABOUT SUITS FOR MALPRACTICE?

Editor, *New England Journal of Medicine,*

The address of Mr. Robert T. Bushnell at the One Hundred and Fifty-Second Annual Banquet of the Massachusetts Medical Society regarding suits for malpractice against doctors, in which he stated, "I have tried one and hope I never have another," leaves one in some doubt regarding his sincerity. For in the same breath he announced, "But I'll try any of them I think are justified against any of you." One would infer that Mr. Bushnell feels it is his solemn duty, however distasteful, to protect the unfortunate patients treated by Massachusetts practitioners of medicine whenever he has reasonable grounds to believe that the physician has been guilty of neglect in the performance of his care of the patient.

It is of some interest to reputable members of the medical profession of Massachusetts to see to what extent other members of the legal profession concur with Mr. Bushnell in regard to similar cases and especially to ascertain if Mr. Bushnell has voiced a current opinion of many of the legal profession as exemplified by statistics.

Since about 1900 there has been a rapid decrease in suits for industrial injuries due to the Workmen's Compensation Laws. Coincidently there has been a rapid increase in the number of suits for malpractice in Massachusetts until recent years. This condition has become so alarming that there are now but two insurance companies that insure the majority of the doctors in Massachusetts against malpractice. It is understood that the reason for discontinuance of this type of insurance by the companies was that it was unprofitable.

It is quite possible that the twentieth century doctor, who is generally conceded to be better trained than his predecessors of the past century, is more neglectful of his patients than the older generations of physicians. But in view of the fact that the legal profession in the state is now so crowded that only about twenty per cent of those examined each year are admitted to the bar, it is interesting to conjecture that new fields of legal practice are being sought. Has the medical profession any protection against unjust suits?

GEORGE A. MOORE, M.D.,
Secretary Plymouth District Medical Society.

RECENT DEATH

CHALMERS — ROBERT CHALMERS, M.D., died on July 22 at his home, 49 Pleasant Street, Woburn, Massachusetts, after a prolonged illness.

He was born in Scotland in 1861 and came to the United States at the age of seven. He graduated in medicine from the Boston University Medical School in 1887 and joined the Massachusetts Medical Society in 1908.

He was active in civic and social affairs having been a member of the Woburn school committee, a director of the Woburn National Bank, consulting surgeon at Choate Memorial Hospital, and a member of the Woburn Medical Association, the Choate Memorial Charitable Association, Mt. Horeb Lodge of Masons, Hugh De Paine Commandery, Aleppo Shrine, the Woburn Rotary Club, the Winchester Country Club, the Vesper Country Club, the Towanda Club and the Middlesex East District Medical Society.

He is survived by his widow, Mrs. Mary A. B. Chalmers and one daughter, Mrs. Marion Fowle.

NOTICES

AN OFFER TO ASSIST DOCTORS

Translations, abstracts and personal readings of German, French, Spanish and Italian medical literature are offered without charge by a physician of Boston.

Applications for this service may be sent to the *New England Journal of Medicine.*

REMOVAL

MIRIAM G. KATZEFF, M.D., announces the removal of her office on August 1 to 270 Commonwealth Avenue, Boston.

REPORTS OF MEETINGS

THE HAMPDEN DISTRICT MEDICAL SOCIETY

The regular summer meeting of the Hampden District Medical Society was held at the Westfield State Sanatorium, Westfield, Mass., on Tuesday, July 11, 1933, at 4:00 P.M., when the members were guests of Dr. Roy Morgan, Superintendent.

Dr. Morgan gave a paper on "Practical Points in Tuberculosis" in which he emphasized the high mortality rate of pulmonary tuberculosis in ado-

lescents and the necessity to employ x-ray to detect early cases.

Dr. Goldblatt presented a short paper on "An Epidemic of Acute Non-Tuberculous Pulmonary Infections Characterized by Rapid Appearance and Disappearance of Pulmonary Lesions."

Dr. William H. Robey, president of the State Society, was introduced by Dr. S. Z. Naurison, district president, and he opened the discussion of the papers.

After discussion a supper was served and there was opportunity for the Society to inspect the Sanatorium.

THE MASSACHUSETTS ASSOCIATION OF BOARDS OF HEALTH

An interesting occasion and an important exposition of care in sanitation were brought together at the meeting, in New Bedford, on July 19, of the Massachusetts Association of Boards of Health. Following a favorite idea of the late Dr. Kennedy of the local board of health, Mr. George T. Glennon and Mr. G. W. Kirschbaum of the present board planned the serving of the collation in the discharging hall of the New Bedford incinerator. Mr. Glennon is the newly elected president of the M. A. B. H. A clambake, brought in trucks from a near-by shore resort, was served in the place normally devoted to the dumping of the loads of garbage and refuse collected in the city.

The hall was airy and well-ventilated, and but for the great iron plates in the floor and the caps of the furnaces, there was not the slightest evidence of the regular use of the place. This is the more interesting in that it has been in service for five or six years and cares for from sixty to seventy-five tons of material each day. Its clean condition was proof of what can be done with proper planning and competent supervision. Mr. A. B. Thresher, superintendent of the plant, has done much more than preserve the sanitary features, for he has relieved the establishment of a purely utilitarian aspect by well-cultivated lawns, skilful disposition of flower beds and the adornment of walls and ramps by ramblers and other attractive species of the rose family.

The meeting was attended by many lady guests, and there was the usual valuable exchange of experiences by the health officers from different sections of the state, with a few short addresses on health topics. The afternoon was devoted to sight-seeing, with visits to the Whaling Museum of the Dartmouth Historical Society, and to Colonel Green's extensive estate at South Dartmouth.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

August 10, 11, 12—The American Delegation to the International Goiter Conference will be held at Berne, Switzerland. Address Dr. James H. Means, Massachusetts General Hospital, Boston, Mass.

August 13-27—The Fifth International Medical Post-Graduate Courses of the Tomarkin Foundation will be held in St. Moritz, Switzerland. The subjects in these courses are Allergic Diseases, Diseases of the Blood, Climatology, Rheumatism and Arthritis and Diseases of Metabolism. Address Chr. Jilli, St. Moritz.

September 11-15—The Twelfth Annual Scientific and Clinical Session of the American Congress of Physical Therapy will be held at the Palmer House, Chicago. Address the Executive Secretary, 30 North Michigan Avenue, Chicago.

September 16—The American Board of Otolaryngology will hold an examination in Boston just prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska.

September 19, 20, 21—The Connecticut Clinical Congress will meet at New Haven. Subjects for consideration: Diseases of the Blood, Diet in Disease, Industrial Injuries and Neuroses. Lectures, Demonstrations, Group Conferences.

September 25-30—The American Congress of Radiology will be held at the Palmer House, Chicago. For particulars write to Dr. Benjamin H. Orndoff, 2561 North Clark Street, Chicago.

September 29 and 30—New England Surgical Society at Boston.

October 9-12—The American Public Health Association will hold its sixty-second annual meeting at Indianapolis. For details on the scientific program write to the American Public Health Association, 450 Seventh Avenue, New York, N. Y.

October 9-13—The Clinical Congress of the American College of Surgeons will hold its twenty-third annual session at Chicago. Headquarters for the Congress will be at the Stevens Hotel.

October 23 - November 3—The 1933 Graduate Fortnight of the New York Academy of Medicine. The theme during the two weeks of intensive study will be Metabolic Disorders. A complete program may be obtained by writing Dr. Frederick P. Reynolds, The New York Academy of Medicine, 2 East 103rd Street, New York City.

October 28—The American Board of Dermatology and Syphilology will hold written examinations for candidates in the following cities: New York City; St. Louis, Mo.; Boston, Mass.; Cleveland, Ohio; San Francisco, Calif.; Philadelphia, Pa.; Chicago, Ill. Information may be obtained from the Secretary, Dr. C. Guy Lane, 416 Marlborough Street, Boston, Mass.

December 15 and 16—The American Board of Dermatology and Syphilology will hold oral examinations in New York City. Information may be obtained from the Secretary, Dr. C. Guy Lane, 416 Marlborough Street, Boston, Mass.

April 16-20, 1934—The American College of Physicians will hold its Eighteenth Annual Clinical Session in Chicago at the Palmer House. For information write Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

July 24-31, 1934—The IVth International Congress of Radiology will be held in Zurich under the presidency of Professor H. R. Schnitz. General Secretary Dr. H. E. Walther, Gloriastrasse 14, Zurich.

BOOK REVIEW

American and Canadian Hospitals. Prepared by the Midwest Publishers Company under the supervision of the American Hospital Association. Published by the Midwest Publishers Company. 1560 Pages. Price $10.00.

This book, edited by James Clark Fifield, is a thorough compilation of facts regarding the hospitals of the United States and its possessions, and the Dominion of Canada. It contains the following data: addresses of institutions, statements as to whether the hospitals are general or restricted to certain types of disease with listings of the diseases cared for and those not admitted, ownership of the hospitals and the form of organization. There are also statistics regarding rates for patients and items of interest to those who are historically minded.

In short, this is a reference book which should be on the shelves of all medical libraries and allied institutions, for there are many who seek the information which it contains.